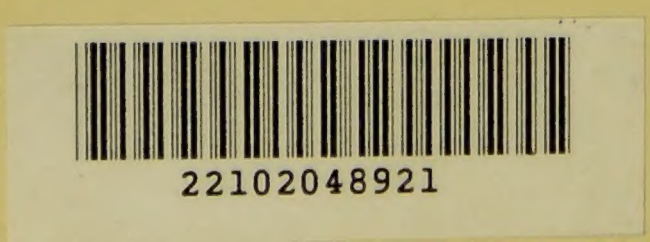
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THE STOMACH


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THE DISEASES OF THE STOMACH

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DISEASES OF THE STOMACH.

SECTION I.

INTRODUCTION AND CLASSIFICATION.

The chief excuse for the existence of a book is its individuality. While it is true that the definitive features of a book are the individual views of the author, the great mass of a complete work on the diseases of the stomach must consist of the results of research gathered all along the course of medical history. A complete book on this subject for the use of students and physicians should contain what of knowledge there is of practical value in the past, what of truth there is in the literature of to-day, and what of information the author may have to contribute. An outline of the evolution of our knowledge of the diseases of the stomach would be a just tribute to the original workers of the past. Possibly narrow and individual conceptions should be rounded with a critical estimate and statement of the opinions of living authorities. But it is our chief endeavor to make this book simple, clear, practical, and complete in useful information. We have consequently decided to have as little as possible to do with history, to omit unnecessary references to literature, and to rearrange the best that others have said and done in unison with what we ourselves have learned during years of special study and practice.

The classification of the diseases of the stomach should be simple and practical, and embody the data of physiology and pathology. It has become the custom, as exemplified in many text-books, to describe as distinct diseases what are in reality only the functional signs of disease or what are merely inconstant accompaniments. Another common error, in our opinion, is the mistaking of a condition for a disease. It will be observed, consequently, that separate chapters are not

devoted to hyperchlorhydria, to hypochlorhydria, to anachlorhydria, to achylia, to gastrosuccorrhea, to erosions of the gastric mucosa, and to dilatation.

The abnormalities of secretion have been classified as chemical types, and described as distinct diseases with a special causation, special evolution, and special medication. But these abnormalities of secretion are signs of disease and nothing more,—be the disease functional or be it organic. The secretory signs are so constant and characteristic in the various anatomical diseases of the stomach that they are of very great positive and negative diagnostic value. But the abnormalities of secretion may not depend on a lesion of the mucous membrane, but on abnormal glandular activity, which may be excessive or diminished. These two dynamic affections of secretion we describe as adenohypersthenia gastrica and adenasthenia gastrica. The occurrence of achylia as a dynamic affection without an anatomical lesion of the mucous membrane may well be doubted, for adenasthenia gastrica rarely, and then only temporarily, advances to this stage represented by complete loss of glandular power. Achylia is a sign of the terminal period of asthenic gastritis, a symptom of atrophic glandular gastritis, and it is sometimes met with in advanced carcinoma.

Gastrorrhea was first described minutely by Parker (1838), although the condition is sometimes known as Reichmann's disease (1882). This symptom-group may occur in the course of chronic glandular (hypersthenic) gastritis, of obstruction to the evacuation of the contents of the stomach, or of myasthenia. The continuous secretion is the result of the retention or of the gastritis or of both; it is a condition, a group of signs and symptoms which may develop in the course of well-known diseases of the stomach, and it never appears to be a primary disease.

Erosions of the gastric mucosa occur in acute and chronic, primary and secondary, gastritis. They may rarely result from cerebral lesions, but are then ordinarily insignificant. We see no good reasons for classifying this epiphenomenon as a distinct disease of the stomach.

The causes of so-called "dilatation of the stomach" are obstruction to the evacuation of the contents of the stomach into the intestines, deformities and adhesions of the stomach, displacements of the stomach, and myasthenia gastrica. The condition has three characteristics: the stomach has become larger, it does not completely empty itself during the twenty-four hours, and it does not retract when it is empty. Some

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authors regard "dilatation of the stomach" as synonymous with enlargement of the stomach: some consider the presence of food in the stomach in the early morning before breakfast as its pathognomonic sign: some think that loss of tone and elasticity is its chief characteristic. The normal stomach may be large or small, and its size has no relation to its motor sufficiency. A stomach which does not empty itself under ordinary circumstances during the twenty-four hours, may be large or small, strong or weak, thick or thin. A stomach which has lost its tone and elasticity and its power to retract when empty is a myasthenic stomach. We shall not describe "dilatation" as a disease; and we deem it best to employ the words "stagnation" and "retention" to denote the degree of motor insufficiency, and to emphasize the fact that motor insufficiency is a functional sign and not a morbid entity. The word "dilatation," like the word "dyspepsia," has no precise meaning, and embodies false notions. These words impede medical progress and should become obsolete.

It is also a mistake, we think, to describe, as so many authors do, all the dynamic affections of the stomach as "neuroses." Some of these troubles are purely muscular. Some of them are exclusively glandular. Some are auto-intoxications. Some are psychic, and some are dependent upon diseases of the central nervous system and of the various important organs of the body. A very few are possibly "neuroses" in the proper sense of the word. All these affections are dynamic. Such is their nature, and there is nothing else palpable in their manifestations. We shall, consequently, devote a section to the dynamic affections of the stomach, and we believe that this classification embodies a doctrine which is in close conformity with the truth.

The displacements of the stomach are frequent. The trouble is a purely physical one. The organ is forced to do its work in an unfavorable position. It may be contended that a displacement is not a disease,—*i. e.*, a morbid process in evolution,—that it is only a condition, a result, an accident. But it is not a condition, which, like "dilatation," forms a stage in the orderly evolution of a disease of the stomach. It is the primitive trouble of the stomach. It has its own causation and proper evolution, and can not be understood apart from them.

The book is divided into six sections. The first section is introductory.

The second section treats of diagnosis and diagnostic methods, the signs of disease being arranged and classified in

the order in which they are obtained during the clinical examination—viz., the clinical history, the physical signs, the functional signs, the bacteriological signs, and the anatomical signs. No chapter will be devoted to the diagnostic value of the alterations of the blood, of the qualities of the stools, and of the properties of the urine. The changes in the blood, the feces, and the urine will be given in the clinical description of each disease. Experience with the more direct and exact methods of investigation renders one very cautious in going back from the changes in the blood and excretions to a particular disease or chemical type. It will not be denied that these deviations possess some confirmatory diagnostic value; but if we attempt to go back to the stomach from the changes in the blood and urine and stools, the conducting thread is soon lost in a network of possibilities. The chance of error is too great.

The third section is devoted to general medication. The principles of therapeutics are discussed and therapeutic methods are described. The first chapter treats of hygienic and physical remedies. The other chapters are based on the information obtained by the examination of the patient, the kind of treatment corresponding with the variety of disease-signs—symptomatic treatment, mechanical treatment, chemical treatment, physiological treatment, and bacteriological treatment.

These three sections constitute the general or first part of the book. The second or special part of the book is devoted to particulars, which represent results and conclusions. It gives what the physician at the bedside discovers with the aid of the methods of examination; and it applies the general principles of therapeutics to the treatment of the particular diseases. The fourth section (the beginning of the special part of the book) treats of the dynamic affections of the stomach, while in the fifth section are described its anatomical diseases. Departing from the classical methods, a clinical description of each disease is followed by a discussion of its diagnostic signs in the order given in the second section, which is the natural method of examination.

The stomach is an organ which rarely escapes undisturbed when the body or any part thereof is seriously diseased; and the diseased stomach may play a part in the causation of disease of other organs. Be the stomach trouble a dynamic affection or an anatomical lesion, a knowledge of its reciprocal relations is exceedingly important. The sixth and last section will describe the vicious circles of the stomach.

SECTION II.

DIAGNOSIS AND DIAGNOSTIC METHODS.

THE object of the interrogation and examination of the patient is the revelation of a disease. This detective work is known as the diagnosis, which forms the basis of a well-regulated plan of treatment.

The diagnosis includes the recognition of the clinical group to which the disease belongs, and the discovery of its nature, stage, associations, and complications.

The diagnosis of a disease of the stomach includes more than a knowledge of the pathological chemistry of digestion. The unhealthy variations of the gastric juice, the perversion of the motor function, the little variations in the quantity of the hydrochloric acid secreted or left free, occupy too much and too exclusively the minds of some practitioners. Our knowledge of these troubles rests on as sure and broad a foundation as does our knowledge of any other class of internal diseases, and includes all the truth revealed by all the methods of investigation that give a new or an additional light. The theory should be constructed out of all the signs and symptoms and not be made dependent on the functional signs alone, or chiefly. Under the glare of novelty the older methods have become too much neglected, and what is new is given too great value and employed too exclusively and indiscriminately.

Each new method of investigation destroys and creates, and modifies profoundly the theory of particular branches of internal medicine. This was the case with the diseases of the chest after the adoption of the method of Lænnec. Such was also the effect of the introduction of the ophthalmoscope, and the laryngoscope, and the cystoscope in a less degree. Each method that reveals the interior of one of the cavities creates a revolution.

Only a few years ago the subjective sensations gave the greatest part of the information utilized in diagnosis concerning the diseases of the stomach. The clinical examina-

tion was nearly resultless, and was confined to the detection of gross abnormalities of size, form, location, density, and sensitiveness. More recent methods reveal the functional power, while the older methods have been made more exact and technical, and the precision and definiteness of all the diagnostic signs have been correspondingly increased.

The diagnostic methods are both special and general. Some of the procedures are such as are employed in the diagnosis of all internal diseases, while others are used only in the diagnosis of the diseases of the stomach.

The direct investigation of the digestive functions is a modern procedure. It has yielded a new set of signs of the very greatest value at the bedside. The stomach-tube has also enriched clinical medicine, by adding to it the bacteriological and anatomical signs of the diseases of the stomach.

To the methods peculiar to the diagnosis of the diseases of the stomach should be added the more common procedures of physical diagnosis and their modifications in the examination of the digestive organs. The modified technic and special devices will receive a careful and exact description in order that the fullest information of diagnostic value may be rapidly obtained.

The revealing signs and symptoms are subjective and objective, or such as are perceived and related by the patient and are detected by the physician. The one constitutes the clinical history; the other the clinical examination.

The clinical history and examination give the data from which, by induction, the diagnosis is drawn. The logical process is an inductive one, but the analysis is supplemented by synthesis, or the orderly arrangement of the salient and valuable points of the clinical history, and the physical, the functional, the bacteriological, and the anatomical signs. After the clinical history and the examination are completed, the symptoms and signs are arranged in the order of their evolution and in their proper causal relations. The symptom-group is next compared with known clinical types and the disease classified according to its clinical expression. Following the thread found in the modification of function and the evolution of unhealthy variations, we arrive in a natural way at the clinical, functional, and anatomical diagnosis. The more exact and complete the data, the surer is the conclusion reached in this way. The result is dependent on the skill of the physician, the truthfulness and intelligence of the patient, and the exactness and efficiency of the methods.

The constructed symptom-group rarely corresponds in

every detail with a special clinical type, but may be the expression of more than one disease. By exclusion the diagnosis is made exact, and the precision essential to purposive treatment is attained. This process is commonly known as differential diagnosis, and is reached by deduction, comparison, and exclusion. The exclusion of the disease suggested by the symptom-group may be dependent on the absence of a cardinal symptom or sign. The result, though based on a negation, is none the less sure.

After the symptom-group is classified and given a particular name, a comparison is then made with the typical clinical form of the malady, and an explanation is sought for the variations of type. The individual and medical constitution are thus brought prominently into view, and a complication or an associated disease may be revealed. If the situation is thus found to be complex, a further problem is the discovery of the relations of the parts, or the associated morbid entities. As a rule, one disease is the primary and predominant one. But the presence of a complication which may be explained as a result is not conclusive of such a mode of origin, and may lead to a false conception of the supposed causative disease. Accidental independent associations are not rare. That a disease may be explained as a complication does not exclude the possibility of its independent existence and development. Two possible explanations of a symptom-group may be equally plausible, and precision in the diagnosis may be impossible. In such cases a supposition should not be mistaken for and defended as the truth.

A disease may have no characteristic sign or symptom-group. The expression may be irregular, indefinite, formless. The deductive method may then be of use.

No mistake is more common than to leave out of consideration the stage of the disease. The symptom-group of an advanced disease is markedly modified by the constitutional state. The organism suppresses or modifies the expression of the disease, and the former salient features are lost. This is particularly true of the final stages of a disease, when the diagnosis is more clearly revealed by the clinical history than by the present state. The death agony so changes the expression as to suggest often the possibility of an erroneous diagnosis. The mode of death is the same in many widely different diseases. The nature of a disease is revealed by its life history.

Diagnosis is a logical method, proceeding by analysis, synthesis, comparison. The mode of reasoning employed has

been described. Diagnosis is also a methodical procedure. In taking the clinical history and making the clinical examination, in order to avoid error and loss of time we should adhere strictly to a general plan. The clinical examination begins with the medical constitution, the strength, and the state of nutrition. Then look over the skin and visible mucous membranes and search for enlarged glands. This is to be done in every case. The next step is the examination of the organ—the stomach, for example—indicated by the clinical history as the seat of the disease. This being completed, we go on to the examination of the other organs, neglecting in no case to examine the liver, the nervous system, the heart and the blood-vessels, the lungs, and the kidneys. An examination of the blood and the urine, and of the stools, should never be neglected; and the female genital organs, if not functionating properly, should also be examined. The presence of a causative or associated disease may make the treatment of a disease of the stomach a failure.

A complete diagnosis of a disease of the stomach is not a simple or an easy matter. In the clinical history are found such symptoms as point to this organ as the location of the trouble. The process of reasoning by which the nature of the disease and its clinical form are detected has just been outlined. But a complete practical diagnosis of a trouble of the stomach includes much more. In the first place, the clinical form should be recognized, when our attention is limited to the predominant characteristics revealed in the manner of the manifestations. The grand clinical character is dynamic, and is either hypersthenic or asthenic. These are the two clinical forms, and are the clinical expression of excessive or of insufficient activity. The diagnosis of the clinical form characterizes in a general manner the treatment, be it sedative, indifferent, or excitant.

The physical examination yields the physical signs, or those obtained by inspection, palpation, percussion, auscultation, inflation, and electric illumination.

The functional signs make clear the actual work done by the stomach, and form the basis of physiological treatment.

The pathological stomach may become the breeding-place of micro-organisms which destroy the food, rob the body of its nutriment, irritate the stomach, and poison the system. The bacteriological signs possess an intense practical interest.

Special therapeutic indications are given by the anatomical lesions, the determination of the nature of which is often facilitated by the anatomical signs.

These signs and the clinical history combined reveal the nature and stage and probable evolution of the disease, and suggest the treatment.

We shall describe them in the following order, which is the one most natural at the bedside:

1. The Clinical History.
2. The Physical Signs.
3. The Functional Signs.
4. The Bacteriological Signs.
5. The Anatomical Signs.

CHAPTER I.

THE CLINICAL HISTORY.

THE revelations of the modern methods of examining the stomach have so deeply engaged the attention of the medical world that the diagnostic value of the clinical history has been almost forgotten. The improved technic of physical diagnosis, the more exact and the more frequent clinical study of the functions of the stomach, the more intimate knowledge of the conditions and the effects of fermentation and putrefaction, the search for anatomical signs in the contents of the stomach, and the recent advances of pathology, have diminished the obscurity which has so long concealed the nature, the genesis, and the evolution of stomach diseases. But notwithstanding the increase of knowledge, notwithstanding the great precision of modern diagnostic methods, the clinical history still maintains all its old utility. The diagnostic value of the subjective symptoms has only been enhanced by our more exact knowledge of their genesis and their evolution.

Unfortunately, many practitioners do not accept this view, but deem the time wasted which is spent in obtaining the subjective history. Many of the constitutional symptoms, it may be readily admitted, are common to all forms of gastric trouble, and are of little value in clinical investigation. Many symptoms, also, are in no fixed relation to the cause, or to the chemical pathology, or to the anatomical lesion; and the complaints of the patient may be so general as to be meaningless. The numerous and different diseases

of the stomach may have a similar group of subjective symptoms. No one symptom is pathognomonic, and even the collections of symptoms, although more characteristic and more suggestive, are hardly less likely to lead to an erroneous diagnosis. Taken singly or combined, the physician may only be able to confirm the patient's statement that he is suffering from a "bad stomach"; and this does not lead very far toward a rational diagnosis or a purposive treatment.

It is quite true that the bundle of sensations or perceptions is often valueless in the naked abstract; and, therefore, a thorough search must be made for the individualizing characteristics and the definitive features.

To search out and keep before the mind the useful subjective symptoms is essential to the proper utilization of the clinical history. The chief importance attaches not to the gross symptom, but to its characteristics. The diseases of the stomach are not silent, and they commonly speak distinctly. Rarely, it may be otherwise. A disease of the kidneys, a compensated heart lesion, a disease of the liver, may long exist without creating a suspicion. This may also happen with a disease of the stomach, and the first revealing sign may be given by the disturbance of the function of some other organ, or the patient may be so accustomed to a "bad stomach" as to consider its existence meaningless, and may consult the physician for some other trouble—insomnia, palpitation, loss of strength, emaciation, cerebral fatigue. But in the large majority of cases the voice is more distinct, and is heard where it is produced. It is quite proper to speak of an ulcer history, a gastritis history, a cancer history, a myasthenia history, a history of neurasthenia gastrica, or a history of any of the clinically well-defined diseases of the stomach; but to be of value, the history must be compiled by the physician out of the material furnished by his purposive questioning of the patient.

The interrogation of the patient demands tact and experience, and a knowledge of human nature and of the "dyspeptic." The "dyspeptic" is a poor student of himself, but a lover of criticism. Unguided, his story consists chiefly of the numerous drugs he has taken, and the many physicians whom he has consulted without benefit. Acrimonious, full of opinions, he remembers vaguely and relates unwillingly the facts concerning his digestive trouble, except in the light of a preconceived opinion. The physician, who is supposed to be familiar with the clinical pictures of the different diseases of the stomach, should, by well-chosen questions, concentrate the

inquiry and the attention on the distinctive and characteristic symptoms and the symptom-groups. The most detailed description by the patient is much less valuable than a clinical history made out on broad schematic lines. Seize at once the most important points in the patient's story, and follow with questions so formed as to define more clearly the relations of the symptoms.

It must not be supposed that the clinical history suffices, alone, to make a diagnosis. The atypical and the secondary diseases of the stomach, without the signs revealed by an examination, are likely to be overlooked. Even when the clinical history of the stomach disease is most typical, the possibility of associated diseases must be remembered, and there may be complications.

The clinical history opens up the way for further and more exact investigation. The interrogation is an introduction and a guide to the clinical examination. The interrogation, however, should not be made the basis of a diagnosis; for such a course may lead to serious errors. The following case illustrates and emphasizes this principle: A patient, aged thirty-eight, nervous temperament, was severely shocked by the sudden and accidental death of his wife. The appetite began to fail, the stomach, after meals, felt uncomfortably full, and the period of digestion was accompanied by restlessness, nervousness, but by no pain. Liquids produced more distress than solid food, and a full meal increased his discomfort and his mental despondency. The patient had lost a few pounds in weight during the four months since the beginning of the attack, but the diet was less than supporting. Myasthenia gastrica was suspected, and the trouble was supposed to be dynamic. The examination revealed the functional signs of carcinoma, and an elongated, ridge-like, hard tumor could be felt just below and to the left of the ensiform cartilage, moving with respiration, but with difficulty fixable on expiration. The subsequent history proved the diagnosis of carcinoma ventriculi to be correct.

The story, as told by the patient, is a bundle of uninterpreted or mistakenly interpreted sensations. The nature of the disease may often be revealed by the manner in which the history is gathered up and related. But, as a rule, neither the nature nor the location of the disease is revealed in this way. The trouble may be in the stomach, the colon, or the gall-bladder, or in some other abdominal organ. The subjective symptoms of a benign or a malignant disease are frequently alike. The clinical history may, but does not often,

suffice; yet it may reveal the location and the origin, and even the nature, of the trouble. To amass information on these three points is one of the objects in questioning the patient.

The first problem is the location of the disease. This, at first glance, may seem very easy of solution; but here mistakes are very often made. It has been often urged that the specialist is too prone to locate disease in the particular organ or organs which he exclusively treats. It has long been the custom to attribute all the gastro-intestinal and the nervous symptoms of enteroptosis to a displacement or a version of the uterus. The eye and the nose have been no less pretentiously forced upon our attention; and the stomach and the intestines, on account of their wide sympathies and relations, are no less apt to monopolize our study.

It is a mistake to direct the questioning solely to the digestive function. There are diseases of the stomach that present no local subjective sign. These diseases may express themselves at a distance, and do their marauding far away from their home—in the head, or the heart, or the lungs, or the kidneys, or the liver, or the sympathetic or central nervous system. There are also diseases of other organs, the only subjective manifestation of which is a disorder of the stomach. The two following cases illustrate what is meant: A patient with a very violent headache came for consultation; there was no syphilitic history, and no malaria, no neurasthenia, no disease of the kidneys, and no uricemia were found. The headache was at times periodical, at times remittent, at times accompanied at night by insomnia and irritable temper, at times most violent during the day; there was no fixed relation to the period of gastric digestion, or to constipation, or to excesses; it was without definitive features, and the patient was not conscious of any abdominal symptoms. The examination revealed a prolapsed myasthenic stomach, with stagnation and fermentation. Again, a young married woman came complaining of very troublesome vomiting. There were no other subjective or objective gastric symptoms. The uterus was normal and non-pregnant. Upon further questioning it was learned that the vomiting was always preceded by coughing, and an examination revealed an incipient pulmonary tuberculosis, the primary disease. Gastric trouble is so commonly a companion of the diseases of other organs that the physician, cognizant only of the abdominal symptoms, is likely to form a false conclusion. Study the whole man, and learn to know him in every part.

The general history should always precede that of the

abdomen. The local phenomena being next studied, and a provisional diagnosis formed, the known effect of the disease of the stomach on the organism should be looked for as a confirmation of the hypothesis. The general history in its grand lines, the local phenomena in every detail and relation, the minute search for the usual effects of the suspected disease, is the natural method of conducting the preliminary examination. The following case illustrates the simplicity of this natural method: A young clerk had always enjoyed excellent health before an attack of typhoid fever, three years ago. Convalescence was slow, but his appetite and occupation forced him to eat ravenously and rapidly. For the two following years he was conscious of having a bad stomach, intolerant of large quantities of fluid and of large meals. Moderation would cause the headache, insomnia, mental depression, and gastric uneasiness and heaviness to disappear. Six months ago, the morning after a heavy, indigestible dinner and several drinks, he had a severe attack of headache, nausea, sour and foul vomiting, which continued for several days. Since that time he has been worse, and vomits large quantities of fluid—sour, brown, sometimes foul, containing food, etc., eaten the day before. During the past six months he has rapidly emaciated; the skin is dry and rough, the urine is scant, feet and hands are cold, there are cramps in the calves of the legs, "the hands and feet often go to sleep," and the nodosities of Bouchard are very plain. These are retention symptoms. The stomach is not empty in the morning, the vomit "three layer," and contains sarcinæ. Test-breakfast, after frequent lavage, gives free HCl. Diagnosis—myasthenia gastrica, with retention.

The disease being located and classified, it is natural that we should next search for its cause. This may be found in the alimentation, the profession, the home life, the family history, or in a past disease. It is specially valuable to know the beginnings of the trouble; for at this early period we are nearest to the cause, and the disease is yet central, undifferentiated, and uncomplicated. The surroundings of the patient at this time, his habits and diet, should be carefully looked into. The patient, if intelligent, may know the cause, and his opinion should be patiently heard.

Such is the value of the clinical history. Such is the proper and natural method of obtaining it and of using it in diagnosis. The subjective symptoms are the manifestations of the disease, and often reveal its nature when their evolution

and diagnostic features are discovered. The clinical history includes:

1. The general history of the patient before the beginning of the present trouble. In his past we may see the shadows of coming events. Diseases of other organs are selective in their influence on the stomach, and may give a clue to the genesis and the nature of the present trouble.

2. The history of the present trouble.

3. The present condition of the patient, which is most satisfactorily revealed, under the guidance of the physician, by his own relation of the history of a single day.

1. The Previous History.—A disease of the stomach, both in its nature and its evolution, is conditioned, among other things, by the cause and the medical constitution. It has been contended that the pathological anatomy bears no relation to the nature of the disease, and that the symptoms are not an expression of the lesion. In neither the primary nor the secondary diseases of the stomach is this contention true. In the sixth section—on the vicious circles of the stomach—it will be made clear in how intimate a relation a secondary disease of the stomach stands to the disease which has caused it. It is on the reality of this relation that the diagnostic value of the previous history depends, and, consequently, much of the information amassed in that section will aid in the detection of the nature of the particular disease of the stomach.

The older clinicians laid great stress on the medical constitution, and it is to be regretted that the diagnostic, prognostic, and therapeutic rules dependent thereon have been allowed to drop into oblivion. The medical constitution, be it inherited or acquired, is the soil in which the disease is to grow, and consequently conditions both its evolution and the character of its results. The physician at the hospital overlooks it; but the physician who knows the individual and the family detects the unique and persistent force revealing itself successively by troubles very various, but one in causation and treatment—this concealed unity of which the arthritic and neuropathic groups of diseases are the simplest examples. What is true of a diathesis is also true of a temperament, and of the new and special adaptations necessitated by disease and environment. One who studies the present condition may detect the functional trouble, the anatomical lesion, and its seat; he will see nothing of this individual peculiarity revealed in the evolution and the mode of expression and

formation of family disease-groups, and demanding long-continued patience, courage, and a treatment of the morbid individual life. The study of the previous history reveals the individual medical constitution, and the probable nature of the secondary and diathesic diseases of the stomach. We come now to the history of the present trouble—its prelude, the manner and date of its beginning, and its evolution.

2. The History of the Present Trouble.—The prelude to the development of a disease of the stomach contains the exciting and often the remote cause. This period of the medical history should be very closely studied, with a view to first locating the cause in the organism, or in alimentation and the material introduced into the stomach. At this time the cause and the constitution together will suggest more definitely the nature of the trouble than at a period more remote from the beginning. These causes, be they mechanical or vital, act through the five functional factors—the nervous system, the circulation and quality of the blood, secretion, muscular action, and absorption.

The age at which the trouble began may suggest the probability or the absence of certain diseases. Cancer is very rare before the middle third of life. Myasthenia is more and more frequent with advancing years. Adenohypersthenia gastrica diminishes from manhood to old age. Ulcer is somewhat rare before twenty. The hypersthenic motor and sensory affections accompany puberty and youth. Anorexia nervosa, nervous vomiting, and gastralgia are most frequent in young girls and middle-aged women.

The symptoms of the beginning often reveal at once the nature of the affection. The acute inflammations, and often the dynamic affections, begin suddenly, with characteristic symptoms. Chronic diseases, when so from the start, begin slowly and quietly. Ulcer may announce itself by a hemorrhage, or by an unheralded perforation, but more frequently by digestive irritation in strict relation with the physical action of the food. Myasthenia is usually insidious in its beginning, but may progress with a bound after an excessive meal. Cancer most frequently begins in the midst of seemingly good health, with symptoms due to, and increasing with, the motor insufficiency, the facility with which the food taken ferments and taxes the peristaltic and evacuating power. The displacements begin with traction symptoms; a completed displacement may long remain symptomless, but it becomes a conscious disease when abdominal tension diminishes or myasthenia gastrica, particularly with fermentation, supervenes.

When all the physiological factors of digestion are more or less implicated, the symptom-group of the beginning is commonly more characteristic than that of a later period. The disease of the stomach may be displayed more characteristically, if not more intensely, in the phenomena which attend its origin.

The evolution of the subjective symptoms may be intermittent, remittent, or progressive. The course of many diseases of the stomach is marked by periodicity and by paroxysms. These qualities may be inherent in the nature of the trouble, the attacks seeming to recur spontaneously or as the expression of accumulated incompetence, each day the organ becoming more exhausted or irritated under the strain of excessive work. In the interval, digestion is unconscious, and an ordinary mixed diet is well borne. Most of the dynamic affections may be periodical or paroxysmal, the attacks often being excited by errors of diet, physical excesses, or mental and moral strain.

In other cases, the course is characterized by remissions. The interval is a period of improvement, but not of health. The disease continues, but is not uniform in the intensity of its expression. Without these exacerbations, the patient would not consult a physician. During the period of remission, the capability of the diseased stomach is better adapted to the work demanded of it. During the exacerbation, this compensation is disturbed by mental, moral, or physical influences, or by bad hygiene, improper diet, and injurious remedies. This is the course most characteristic of the anatomical diseases of the stomach. Both the remissions and the exacerbations may be due to "little accidents." Another organ may become insufficient, and include the stomach in the vicious circle. The offending matter may be vomited, or may be evacuated into the intestine. After a hemorrhage, the subjective symptoms of a gastric ulcer may subside.

But, be the course intermittent, remittent, or continuous, progression in spite of proper treatment is a suggestive evolution sign. The obstinacy is due to the character of the lesion itself. This malign tendency is most constant in carcinoma, the gross subjective symptoms of which may often be controlled or mitigated by judicious treatment, but each day the disease burns more and more of the albumin of the body, and brings the patient nearer to the grave.

The evolution of a disease of the stomach may be slow or rapid. The clinical period of cancer does not last longer

than twelve to eighteen months. The dynamic affections may continue for years, or may suddenly begin, run a rapid course, and just as suddenly end. The constitution, the moral atmosphere, and the mode of life have much to do with the persistence of these troubles. Acute gastritis and ulcer may soon heal, but the displacements and anatomical diseases of the stomach have a tendency to persist. Expectant treatment has no ally in an inherent tendency to get well, and the diseases of the stomach recur more frequently than those of any other organ.

Some diseases are also accompanied by diminished power of the organism to repair its losses, or to recuperate after trifling accidents. In ulcer, the effects of a hemorrhage may rapidly disappear, because the disease is a local one, and the motor power of the stomach is good. The emaciation is due to voluntary starvation, unless the food be lost by vomiting or by retention from pyloric swelling or stenosis. After a hemorrhage in cancer, the patient sinks more rapidly under the auto-intoxication and the inanition. This diminished recuperative power also characterizes the uncompensated diseases of the stomach.

The evolution of a dynamic affection of the stomach may end, eventually, in a destruction of a large part of the noble elements of the organ. The different stages are links of one chain. Adenohypersthenia gastrica may terminate in gastritis and atrophy, and the early hypersthenia may develop into the most complete asthenia. The symptom-group is correspondingly changed by the substitution of the signs of depression for those of irritation. Pain is no longer complained of, and vomiting may occur only as a result of overdistention, or as a pressure symptom reflected from an adjacent abdominal organ. In cancer, for example, vomiting is rare in the beginning, may become uncontrollable, and then disappear entirely as the disease progresses. Retention marks a turning-point in the history of a disease of the stomach. The delay in evacuation becomes greater and greater, until finally the stomach never empties itself completely; this condition robs the system of its nutriment, and forms a persistent culture-soil for micro-organisms, since the stomach always has something in it. The clinical picture becomes modified by the little accidents without the natural evolution being interrupted. The morbid unity is preserved beneath the play of expression. These changes of type develop in so orderly a manner as to be of value in determining the nature of the anatomical disease following the dynamic affection.

The effects of treatment on the evolution of the disease may reveal its nature. In cancer, the treatment, however purposive, only relieves the gross symptoms of stagnation or retention. The effect of alkalies is of differential value. If large doses of the alkaline waters or drugs give relief, adeno-hypersthenia (excessive secretion) is the most probable functional disturbance; similar doses in myasthenia make matters worse. Lavage is useful chiefly where there is stagnation or retention, be it with or without a trouble of secretion. Only the mild dynamic affections are rapidly influenced by hydrotherapy. The magical effects of a properly-fitting bandage are only obtained in displacements, before myasthenia develops. An ulcer-cure may reveal quickly a suspected ulcer, or, rather, may confirm the probable diagnosis. The therapeutic test, be it positive or negative in its influence on the evolution of the disease or of the symptoms, is a valuable aid in the diagnosis of difficult cases.

The state of nutrition is a revealing sign of great value. The dynamic affections, in spite of their violent expression, may run their course under the appearance of perfect health, without cachexia or loss of weight or strength. The health is preserved in its bloom. But in the painful diseases of the stomach, emaciation may be so pronounced as to present a special cachectic appearance. This is the rule when nutritive waste remains active, instead of sharing—as in anorexia nervosa—in the restraint of other functions. The emaciation of cancer is progressive. In chronic gastritis the weight is preserved so long as the motor power of the organ is intact and the intestines are healthy. Should emaciation occur, in spite of a suitable and sufficient diet, there is stagnation, or retention, or a complication. Inanition, with its emaciation, weakness, and often discoloration of the skin, may be due to a motor insufficiency or to frequent vomiting. The food is not utilized, but destroyed or lost. Rarely can it be said that the emaciation is the direct expression of the disease. But this is not all. The emaciation may be due to an unsuitable or a starvation diet. Nothing is more common than to see large quantities of fluid—soups, drinks of all sorts—poured into a myasthenic stomach, with the result of increasing the stagnation, or of producing retention. As soon as retention begins, emaciation sets in. The diet may be suitable to the disease, but insufficient to maintain the balance of nutrition. A diet which produces emaciation should be temporary, purposive, and imperatively demanded; otherwise it is injurious.

3. The Present Symptoms.—The history of the present trouble being thus clearly marked out in its main lines, the object of further questioning is to obtain a knowledge of the present subjective symptoms. The special history has reference to the past and to the present—the genesis and the evolution on the one hand, and the present condition or stage of the disease on the other. We have, then, the past symptoms and the present symptoms.

To obtain a knowledge of the present subjective symptoms in their natural relations and characters it is a very good plan to ask the patient to tell the history of an entire day, beginning in the morning with the empty stomach, and recounting the symptoms as they appear—what he did, what he ate and drank, and what he felt. The symptoms should be grouped according to their existence during the period of digestion or of functional rest; and their relations to the evolution of digestion, more particularly of the chief meal, should be carefully noted.

For the stomach, the periods of activity and repose are quite clearly marked physiologically; but it should not be forgotten that the intestines also begin work soon after a meal. There is great danger of confounding intestinal and gastric symptoms. On account of the mode of suspension and fixation of the intestines, traction symptoms are often referred to the epigastrium. The colon passes above the umbilicus, and the duodenum, with coils of the small intestine, is found in the epigastric region. Many so-called stomach pains, occurring during the period of gastric digestion, are located in a diseased colon. The subjective symptoms due to diseased intestines, and occurring when the stomach should be at rest, may be referred to this organ by the patient. The interpretation of the patient should not be implicitly accepted, and we should be sure that the symptoms are gastric. The neglect of this precaution may lead to error.

The adoption of a special set of questions as a routine is best, in order to assure a well-grouped picture. During the patient's recital of the subjective history of the day, our questions should bring out all the diagnostic features of the important symptoms.

In this history of the full day we learn the diet—its composition, quality, mode of preparation; the relation of the symptoms to the taking of the food, to the physiological action of the aliment and drinks, to the periods of functional activity and repose of the organ, to the periods of activity

and of repose of the body. We note also the time of appearance and the evolution of the symptoms in relation to the process of digestion; the time of appearance and the evolution of the symptoms as regards exercise, work, and repose.

The condition of the patient before breakfast may be of diagnostic value. The most characteristic symptoms of some diseases appear at this period—such as the early morning nausea, the vomiting of mucus and bile in alcoholic gastritis, and the morning sickness of pregnancy. In gastric stagnation and retention, in morbid sensibility of the sympathetic centers, the person arises tired, and in a state of irritability and of mental and moral depression. If there be no retention and no neurasthenia, the period before breakfast is the best during the day. To make these rules valid, an intestinal trouble should first be excluded. The morning nausea, vomiting, vertigo, headache, and diarrhea, are frequently the symptoms of an intestinal disease.

The traction sensations in the displacements begin with rising, continue throughout the day, and subside with the evening repose.

The digestive symptoms may or may not begin with the breakfast, depending on the nature and the stage of the disease and the physiological action of the food. This small meal may be without bad effect, and may be taken as a rough measure of the severity of the trouble, according as the action is positive or negative. The digestive symptoms are more marked after a full meal, and may occur during the period of the rise, persistence, or fall of hydrochloric acidity.

The most common immediate symptoms are the variations of general sensations. The patient becomes conscious of digestion. There may be only a feeling of unrest, discomfort, heaviness, pressure, as in gastric neurasthenia, gastritis, or cancer, or the uneasiness may be the beginning of a paroxysm of severe pain, which may slowly develop side by side with the evolution of secretion,—as in hyperchlorhydria,—or which may become suddenly violent, as in hyperesthesia, or in a fresh or a sensitive ulcer. Whether these severe pains are nervous or symptomatic of ulcer, they are most frequently encountered in anemic girls. Vomiting as an immediate symptom, unpreceded by nausea or pain, is a reflex motor nervous affection. When developing as a sequence of pain or nausea there is usually a pathological state of the mucous membrane. Nervous vomiting is most frequent in women. The motor disturbances of the stomach, and also its secretory

troubles, are very frequently due to the setting in motion of a diseased intestine from which the reflex proceeds.

During the stationary period of hydrochloric acidity, two characteristic symptom-groups may present themselves—the one is accompanied by severe pain, the other by a sensation of heaviness, of pressure, or of unrest. To the one belong gastric neurasthenia, chronic asthenic gastritis, myasthenia; to the other, superacidity, ulcer, cancer, and hypersthenic gastritis. Fermentation may complicate either form. In the asthenic group, to the unrest, pressure, weight, mental and moral depression, are added meteorism, acid regurgitation, and pyrosis, or heartburn, if fermentation be present. The symptoms are very variable in degree and in duration. The hypersthenic form is accompanied by severe pain, and by other signs of reaction and irritation. The burning sensation, heat, and drawing together of the stomach develop into a cramp, which may become intolerable. The paroxysm is of variable duration, and ends with the passage of the contents into the duodenum, or with their removal by vomiting. In this form there may be in the stomach a moderate degree of fermentation, which may be the cause of the state of irritation; though if the stomach be cleansed and a test-meal given, the quantity of organic acids is very small. In the majority of these cases the fermentation and decomposition are in the intestines, and the gastric symptoms are due to auto-intoxication.

The gastric symptoms occurring only during the period of decline may be motor, secretory, or sensory. In the first there is stagnation or retention; in the others there is abnormal secretion or fermentation.

Many of the gastric symptoms mentioned in the one-day history related by the patient are common to several different diseases of the stomach. Individually and out of their proper relations they possess very little diagnostic or differential value. But some of these symptoms occur only in particular diseases of the stomach, and often have features so characteristic that their genesis is made clear. The meaning of the more frequent of these symptoms, particularly when endowed with special qualities, will now be briefly discussed.

Dysphagia.—In dysphagia the trouble may be in the power of swallowing or in the permeability of the esophagus. The loss of power to swallow is due to paralysis of the muscles of deglutition; paralysis of the esophagus alone is not known. The obstruction of the esophagus may be due to a defect in development, to certain diseases of its walls, and to pressure

exerted at some point along its course. The only two diseases of the stomach that produce trouble in the swallowing of food, or, more explicitly, that interfere with its passage into the stomach, are spasm and organic obstruction of the cardia.

Heartburn.—Heartburn is due to excessive acidity of the contents of the stomach, to hyperesthesia of the cardiac region of the stomach, or to the regurgitation of the irritant contents of the stomach into the lower part of the esophagus. It was long supposed to be a pathognomonic sign of fermentation in the stomach, and it is indeed most frequent in fermentation accompanied by the formation of organic acids in considerable quantities. When due to fermentation, it is most commonly excited by butyric acid, the other forms of fermentation rarely producing it. It is common in myasthenia, cancer, and in the other diseases of the stomach accompanied by stagnation or retention. Heartburn may also be due to excessive hydrochloric acidity. When not produced by eating rancid food, it is a sign of either fermentation or of excessive hydrochloric acidity, hyperesthesia and the regurgitation of the normal contents rarely causing it. Its frequent presence in a particular case possesses some negative value, and aids in the exclusion of the diseases of the stomach in which fermentation and excessive secretion do not occur.

Uncomfortable Sensations.—A sensation of weight and fullness in the stomach is a very common symptom. The sensation of traction is also frequent, and the three sensations are often present together, and confounded by the patient. These sensations possess no definite diagnostic value. In some cases they are present for only a short period after the meal; in others, their evolution is progressive throughout the period of digestion. Without possessing an absolute meaning in such a case, the sensations of weight and fullness which are often associated indicate varying degrees of myasthenia. The symptoms often disappear when the sleepy muscle awakes to its work, and the stomach gets rid of its contents by absorption and peristalsis. When the symptoms persist through the unusually long digestive period, the atony is more pronounced. Gastric neurasthenia is frequently accompanied by the same sensations, which may be due in part to the increased sensitiveness of the nerve-centers during the period of physiological activity. The patient becomes conscious of sensations connected with the state of the gastric muscle, which in health go unperceived. The neurasthenic sensations are not so intimately connected with the quantity and the

quality of the food as are those of myasthenia and gastritis. These sensations may persist during the period of physiological rest. There is then prolapse, a marked myasthenia, or even retention. When the abdominal wall is atonic, the symptoms are frequently due to coprostasis. In both coprostasis and prolapse of either the stomach or colon, marked relief is at once given by support. These sensations, in a general way, point to myasthenia, gastritis, or to displacement of the stomach or colon; they are due to the overdistention of the muscular layer, to the stretching of the nerves or the supporting ligaments, to compression of the sensitive nerves against adjacent organs, and they are intensified by neurasthenia.

Pain.—Abdominal pain is a symptom of great diagnostic value, provided its qualities and associations be clearly defined. Pain in the abdomen only indicates the probable location of some sort of disease in this region; it is a revealing sign of a particular disease only when its individuality is exactly and clearly defined. The first problem is to fix its location in the stomach; and then, through its qualities and associations, we can reason back to its particular cause.

Neuralgia or rheumatism of the abdominal wall may be mistaken for pain in the intestines or in the stomach. In muscular rheumatism the pain is relieved by immobilization, excited by muscular contraction, and is often accompanied by a slight rise of the temperature. The muscle is then often contracted. Neuralgia follows the course of the nerves, and has its painful points. Neither are in any relation to the taking of food, although neuralgia may be increased, or even relieved, by the activity of the stomach; the neuralgia is then associated with the other signs of neurasthenia. Pressure may locate the trouble; in neuralgia, the skin is sensitive; in rheumatism, deep pressure with the flat hand does not increase the pain, which is then also diffused, corresponds in distribution to the muscle or the muscles affected, and is never circumscribed and limited to a very small area. The pains of neuralgia and rheumatism are continuous, and are subject to exacerbations. Both may be associated with pains in other regions.

A pain located in the stomach is not always due to a disease of this organ, which is often the seat of referred pain. Examples of referred pain are met with in the gastric and the intestinal crises of tabes, subacute myelitis, compression myelitis, and aneurysm of the abdominal aorta. The pain of the central lesion—of irritation by pressure—is referred to

the peripheral area of distribution, in accordance with a well-known law. In all cases of severe abdominal pain care should be taken to exclude these causes before attributing the pain to a disease of the stomach.

A pain which has the stomach for its seat, and which is not produced in the foregoing manner, is often erroneously accepted as a sign of a local disease of this organ.* Pain in the area of distribution of one branch of a nerve may have its origin in the periphery of another branch of the same nerve. This is especially true of the pneumogastric with its innumerable connections with the abdominal sympathetic. The pain is often in the stomach, when the disease is in the intestines or elsewhere. It is a good rule to suspect the colon whenever the stomach is the seat of a so-called colic.

When hepatic colic is attended by its typical symptoms, such as the paroxysmal pain, icterus, distention of the gall-bladder, peritoneal friction, fever, and enlarged or tender liver, and when calculi have been discovered in the stools, there is no difficulty of diagnosis. But in the atypical or abortive attacks, the differentiation may be more difficult. There are, however, many minor signs which may reveal the origin and location of the pain.

The pain of biliary colic is located over the gall-bladder, at a point three fingers' breadth to the right, and the same distance above the umbilicus. Here the pain begins, and radiates to the right into the back and shoulder, and to the left into the epigastrium, and over the whole abdomen. Pressure over the liver is painful; pressure over the uncovered area of the stomach, if care be taken to avoid the left lobe of the liver, is not so. The dorsal painful-pressure point is to the right and not to the left of the vertebral column, as is the rule in ulcer. Hepatic colic may be excited by a dietetic error, and is not altogether independent of digestion. But it does not appear with regularity after each meal, or necessarily during the digestive period, and is not dependent on the physiological action of the food. The paroxysms of hepatic colic are separated by intervals during which all foods are equally and perfectedly digested. The causes which excite the attack are not so palpable as in the diseases of the stomach, but the attacks seem to recur almost accidentally. In hepatic colic there is no typical alteration of gastric secretion, but the vomit is often superacid. The diagnosis may be very difficult when both a painful hepatic disease and a gastric disease are at the same time present, without char-

acteristic symptoms. The attacks of hepatic colic are rarer, and recur irregularly, and often suddenly, without being accompanied by the emptying of the stomach. Nor is hepatic colic ever relieved by food, alkalies, or fluids. Intestinal colic, beginning in the hepatic flexure of the colon, may simulate hepatic colic.

A large number of diseases of the stomach are painless, or can run their course without distinct pain. This is the case in chronic asthenic gastritis, in many of the dynamic affections involving only the motor functions, and in the milder cases of myasthenia. Under these conditions notable pain is exceptional. Consequently, when pain is a prominent symptom, these diseases may be excluded.

In other diseases the pain is an accident. Such is the case in myasthenia, and in the milder cases of retention, when the pain is the result of fermentation more dependent on the quality of the diet, or on an excessive quantity of food, than on the condition itself. These accidental pains are the result of overdistention, or of local irritation by the products of fermentation.

In other cases the pain is periodical, and it then appears either during the period of functional activity, or is independent of digestion. In adenohypersthenia the pain develops with the increase of free hydrochloric acid, appears after each meal, and is relieved temporarily by the ingestion of proteids (milk, meats, eggs) and water. In hyperchlorhydria pain is not always present; when it is present, there must be an increased irritability, in addition to the excessive formation of the hydrochloric acid; or there must also be hyperesthesia of the mucous membrane.

Gastric pain may be *continuous*. Such is the rule with ulcer and advanced cancer. The lesion may allow no periods of entire freedom from pain, while a combination of circumstances increases the pain during the period of digestion. The pain of pyloric carcinoma is often severe, but is even more persistently severe on account of the lactic acid formation and of fermentation. Of all the diseases of the stomach, ulcer and pyloric cancer are the most constantly painful.

Gastric pain may be entirely independent of abnormal nerves. The pain is then due to compression by muscular contraction, when the pain is acute, and subsides with the relaxation of the muscle; or it is due to the overdistention with gas, in which case it is dull, aching, heavy, and disappears with the escape of the gas. The pain is the expression of the motor affection.

The favorite site of gastric pain is the pyloric region, even when the disease is not confined to this part of the stomach. If this symptom be carefully studied as regards its genesis, evolution, duration, intensity, location, diffusion, relations to digestion and to the period of functional repose, to the quality of the food, to the repose or activity of the body or the mind, there is no other gastric symptom that possesses so great a diagnostic value. Its distinctive features in the various painful diseases of the stomach will be found in the fourth and fifth sections of this volume.

Nausea.—Nausea is a frequent symptom of the diseases of the stomach. It sometimes accompanies a dynamic affection of the stomach, in which case, on close investigation, the cause will often be found to be a reflex or an intoxication expression of an associated intestinal disease. In acute and chronic gastritis nausea may appear after each meal; often also during the night, or early in the morning. In the gastric troubles of the anemias it is also frequent. Nausea may occur when the stomach is empty or during the period of gastric digestion. In its nature it is a subjective sensation of disordered peristalsis reflexly produced through the vasomotor nerves, or it is an expression of auto-intoxication, or of irritation of the stomach or the intestines. The symptom is most frequent in the anatomical diseases, and in motor troubles accompanied by fermentation and putrefaction. In cancer it is a more frequent symptom than vomiting. It should be noted whether the nausea is constant or occurs only when the stomach is empty, or only during digestion, and particularly with what symptoms it is associated. It is a rare symptom in many of the diseases of the stomach, and is often present when the stomach is not diseased.

Flatulency.—Flatulency, or the gaseous distention of the stomach, is a frequent symptom. It may be produced in two ways: by the presence of an abnormal quantity of gas in the stomach, or by myasthenia gastrica.

Myasthenia is the most frequent cause of flatulency, the atonic wall yielding to the natural expansibility of the contained gas. This form of flatulency may be quickly relieved by a few eructations, which, however, it may be very difficult to produce voluntarily. There may or may not be an excessive quantity of gas in the stomach, but gas readily accumulates in the myasthenic stomach.

The gases found in the stomach are swallowed, generated in the stomach by chemical decomposition or by fermentation, or are regurgitated from the intestines. Flatulency

may consequently be a sign of excessive swallowing, or too much saliva (carbonates) may find its way into the acid stomach, or there may be too great a quantity of carbonates in the food and drinks. Effervescing drinks also furnish a quantity of gas. The gas regurgitated from the intestines may be formed by fermentation and putrefaction or by the action of the acids of the chyme and of the organic intestinal acids on the carbonates of the pancreatic and intestinal juices. Carbonic acid gas may also be set free in the stomach from the pancreatic juice when it flows back into the stomach. But the most frequent cause of the excessive formation of gas in the stomach is fermentation. So-called gastro-intestinal respiration may be entirely neglected. The excessive quantity of gas may accumulate in the stomach in spasm and obstruction of the pylorus. Clinically, daily recurring flatulency (not belching simply) is a sign of either myasthenia, pyloric spasm, or of obstruction, or of gas-forming fermentation. This guide is more trustworthy if care be taken to keep the bowels freely open and to exclude effervescing drinks and carbonates.

Regurgitation.—In regurgitation the contents of the stomach are brought up into the mouth, usually involuntarily and without effort. When the stomach contents are returned to the mouth to be again masticated and swallowed, the condition becomes a distinct affection known as rumination.

Regurgitation occasionally occurs as a mere accident or episode of normal digestion, particularly after a heavy meal. Pathologically, regurgitation may be a symptom of a disease of the stomach, or, when it is habitual, constitute an idiopathic dynamic affection of the stomach, described in the fourth section of this volume.

Regurgitation is most frequent during the period of digestion, but may occur during the period of repose. Occurring during the period of repose of the stomach, the regurgitated matter is usually insipid or slightly acid, and it consists of saliva mixed with exudation and mucus from the gastric mucous membrane, and sometimes also it is composed in part of the unevacuated remnants of the previous meal. This form of regurgitation is most frequent in myasthenia with stagnation, and in mild cases of gastritis.

Digestive regurgitation is produced by overdistention of the stomach, and by excessive hydrochloric and organic acidity. It is most frequent in myasthenia and in gastritis or gastric irritation. If the flavor of the regurgitated matter is both bitter and acid, so as to set the teeth on edge, hydrochloric superacidity is most likely the cause. If only bitter, the

taste may be due to altered bile or to peptones. If the regurgitated matter both tastes and smells sour, there is probably fermentation. In cancer of the esophagus or stomach, and in esophageal retention, it may be foul. It should not be forgotten that regurgitation may be esophageal, and the matter consist of food and saliva collected above a constriction, in a pocket, or above a spasm or obstruction of the cardia.

In pyloric incontinence, myasthenic retention, gastroptosis, neurasthenia gastrica, and in many of the dynamic affections, regurgitation rarely or never occurs, and the symptom possesses a negative diagnostic meaning.

Vomiting.—Vomiting may be a symptom of a large number of diseases, and does not necessarily imply that the stomach itself is abnormal. Vomiting is almost as frequent a symptom of the diseases of the intestines as of the diseases of the stomach itself. All the forms of vomiting should be carefully separated from the vomiting symptomatic of a disease of the stomach. Nervous vomiting as a morbid entity is rarer than is commonly supposed. For the discussion of this symptom, and of hematemesis, the reader is referred to the chapters on Nervous Vomiting and Ulcer, respectively.

Appetite.—The variations of the appetite, to be of diagnostic value, must be the expression of a disease of the stomach. Fatigue, the change from an active to a sedentary life, a hot climate, old age, monotonous diet, moral strain, physical pain, are all accidental causes of a diminished appetite. The appetite may be very variable, and not in relation to the state of the stomach, but to that of the brain. The general or local cry for food, or the protest against it, is too composite to be utilized to point to the location and the nature of the disease except under a particular condition. The variations are not infrequently due to the coating, the laying bare, or the alteration of the nerves of taste, in the diseases of the mouth. The appetite is influenced by the environment, the mental and moral conditions, the state of nutrition, and by many organic and general diseases, but particularly by intestinal auto-intoxication and by severe pain. The appetite, however, when associated with symptoms pointing clearly to a disease of the stomach, becomes of use in diagnosis.

A false hunger may appear at the end of the period of digestion, and is then due to the continuance of secretion after the stomach is empty.

In all the secondary and the complicated diseases of the stomach the character of the appetite possesses little diagnostic value, and does not indicate the form of trouble present.

The appetite also does not vary with the changes of secretion. In simple subacidity without fermentation the appetite is normal. In simple superacidity, however, the appetite is normal or increased. In the dynamic affections, not assuming a fixed character like bulimia and anorexia, or in the anatomical diseases growing in a neuropathic soil, the appetite is often rapidly variable, extremely perverted, or easily changed by mental impressions or by a few mouthfuls of food. But these are only presumptive signs that demand control, and possess no characteristics which can be considered as certain or absolute. The rules admit of many exceptions because of the many associations and the wide relations of this special sensation.

Anorexia, when the expression of gastric trouble, is either nervous or organic. The nervous form occurs in the neuropathic girl, is commonly complete for all food, and gives no concern to the patient. Long-continued anorexia, due to organic disease of the stomach, is a very grave sign. The patient makes every effort to overcome it, but without success.

In cancer, and in other anatomical diseases attended by bacterial decomposition of the food, the appetite is diminished and may develop into disgust for particular classes of food. The appetite grows less and less as the intervals when the stomach is empty become shorter and shorter. The food against which the appetite is directed belongs to that class which is undergoing fermentation or putrefaction. In ulcer the appetite is maintained, though the quantity of food eaten may be markedly diminished on account of pain or the fear of pain associated with the entrance of food into the stomach. In the latent form of ulcer the appetite is, as a rule, uncommonly good.

Thirst.—Thirst is a less valuable diagnostic sign. It is augmented in fermentation, and in diseases attended by a marked diminution of absorption. An increased desire for fluids is often present, also, in the secondary gastritis of both alcoholism and Bright's disease. Thirst is also a sign of poverty of the fluids of the body, caused by increased elimination, by loss in hemorrhage, and by vomiting. In supersecretion, the desire may be almost irresistible. Thirst is sometimes diminished in the nervous affections.

The gastric symptoms which have been thus far described are primary, being generated and expressed in the stomach itself. But there are also secondary symptoms, which are very frequent, and are very valuable guides in the search for

the existence of a stomach disease. The secondary symptoms may be the only manifestations, the primary symptoms sometimes being altogether absent.

The secondary symptoms are generated in three ways: some of them are reflex and nervous; some are due to gastric auto-intoxication; others are the result of inanition.

Emaciation and loss of strength are symptoms of only a few of the simple diseases of the stomach. The inanition is often wholly due to an insufficient diet, the little that is eaten being well digested and utilized. This form of inanition is seen in anorexia nervosa, in ulcer, and in the other painful diseases of the stomach. The insufficient diet may be self-inflicted by the patient, or mistakenly or properly prescribed by the physician for a definite purpose. In other cases, the inanition is the result of excessive vomiting, or of the malignant nature or the advanced stage of an anatomical disease. There is no doubt that long and severe suffering may produce emaciation and loss of strength, a fact well expressed by the phrase "the disease is wearing me out." But the most frequent causes of inanition of gastric origin are obstruction of the pylorus and myasthenic retention. The food is imperfectly digested, is often periodically lost by vomiting, and undergoes fermentation in the stomach, and putrefaction and fermentation in the bowels. The pernicious effects of auto-intoxication result, and the intestines, being disordered by the vicious chyme, are unable to establish digestive compensation. Inanition is also marked in uncompensated gastric atrophy. Whenever emaciation and loss of strength are associated with a disease of the stomach, we should first ascertain whether a sufficient quantity of food is being eaten and retained, and whether the intestines are healthy. If such be the case, marked inanition is most likely due to carcinoma, or to some of the causes of gastric retention. Long-continued retention and cancer inevitably produce inanition. Anorexia nervosa, vomiting, ulcer, and hypersthenic gastritis may result in inanition. The other diseases of the stomach do not directly affect nutrition. Consequently, the symptom has both a negative and a positive diagnostic value.

The reflex nervous symptoms are very numerous, and are dependent for their existence and their intensity on the sensibility of the nervous system and on the irritation of the gastric nerves. The irritable weakness of the nerve-centers is produced by the numerous causes of neurasthenia. The gastric nerves are irritated by the contents of the stomach-

products of fermentation, irritant foods and drinks, prolonged digestion, as in retention, stagnation, and intemperate eating —by an anatomical lesion like ulcer, hypersthenic gastritis, cicatrices, adhesions, and cancer, and by the nerve stretching incident to developing displacements of the stomach.

The gastric irritation is transmitted by the vagosympathetic to the spine, to the medulla, to the brain, and directly also to the heart, lungs, and intestines. In this manner the functions of these parts become disturbed.

Gastric is much less frequent than intestinal auto-intoxication, because in the stomach putrefaction is rare, and absorption is slow or absent. A healthy liver is usually able to protect the system against gastric intoxication, but the functions of the liver are, unfortunately, often insufficient. The barrier against gastric intoxication is then removed, provided the poisons are there formed in sufficient quantity. That poisonous doses are formed in the stomach is by no means certain.

It is well known that chronic putrefaction is rare in the stomach. Sometimes hydrosulphuric acid is formed in benign retention, but in no greater quantities than occurs in the intestines without giving rise to intoxication. It is certainly true that acute gastric putrefactive auto-intoxication does occur, but usually only when the poisons have been formed before the food enters the stomach. In every case of chronic putrefactive poisoning that has come under our observation it has proven to be of intestinal origin, even where associated with gastric retention. It would be rash to deny that tox-albumins are formed in the pathological stomach in sufficient quantities to produce auto-intoxication, but a theory built on such a supposition is based on our ignorance.

Gastric fermentation, however, is very common, but the gases and the organic acids formed by fermentation are not very poisonous. The most injurious are the fatty acids. The others have only a local effect.

The secondary symptoms are very numerous, but are of very little utility in the diagnosis of a particular disease of the stomach. Certain mental symptoms, disorders of sensation, disorders of the special senses, disturbances of the heart's action, cough, dyspnea, vertigo, drowsiness, stupor, coma, headache, neuralgia, tetany-like cramps, extreme and intermittent muscular weakness, etc., may be due to a disease of the stomach. These symptoms are described in the sixth section.

CHAPTER II.

THE PHYSICAL SIGNS.

The physical signs are those obtained by the physician through the special senses, and include inspection, palpation, percussion, inflation, auscultation, and electric illumination, aided by special devices. These objective signs reveal the physical deviations from the normal, and instruct us concerning the size, the form, the position, the distensibility, the muscular activity, the neoplasms, and the sensibility of the stomach, as well as some of the physical properties of its contents.

The physical examination of the stomach is a very old procedure, but during the past decad it has been improved in technic, and the precise meaning of each sign has been sought with tireless energy. The physical examination is no less important than the more modern functional exploration, and has the unique advantage of being universally applicable.

The physical examination of the stomach is based on a knowledge of its practical anatomy, and the clinician should be well grounded in this branch. Many of the finer anatomical details may properly be neglected at the bedside, and only those which have a practical bearing need here be recalled to mind.

The digestive tube is a permeable canal, developed from the tegumentary layer, beginning with the mouth, and curling irregularly through the body it ends with the rectum. The mouth and esophagus, and their connecting cavity, the pharynx, lie above the abdominal cavity, which extends from the under surface of the diaphragm to the rim of the true pelvis. The rectum, the lowest division, lies in the cavity of the true pelvis; the other anatomical divisions—the stomach, duodenum, jejunum, ileum, cecum, colon, and the sigmoid flexure—are situated in the abdominal cavity.

Fixed points on the abdominal wall should be taken as the surface landmarks to be used in the investigation of the physical changes and relations of the abdominal organs and viscera. The division of the abdomen into regions, by arbitrary and imaginary lines, is not free from objections. We shall take the easily accessible points of the skeleton and the umbilicus as the basis of abdominal localization. Of these, the umbilicus is the only movable and variable point. In the

perfectly normal abdomen, the umbilicus is in the median line in front and on a level with the tip of the spine of the third lumbar vertebra, a little below the center of a line joining the ensiform process and the symphysis, and about one inch above a line drawn across the abdomen between the highest points of the iliac crests. Thus is found the normal umbilicus or umbilical point.

The skin over the epigastrium is supplied by the sixth and seventh intercostal nerves, the region about the umbilicus by the ninth and tenth, and the iliac regions by the upper lumbar nerves. The lateral cutaneous nerves (branches of the lower seven dorsal and two upper lumbar nerves) supply both the skin and the muscles of the abdomen. Pathological irritation of these nerves contracts the abdominal muscles and produces pain about the umbilicus.

The sensory nerves of the abdomen are in communication with the thoracic sympathetic ganglia, from which arise the greater and the lesser splanchnics. It is through these nerves, which are connected with the cord and the abdominal ganglia, that the skin becomes very sensitive, and the abdominal muscles become contracted, in the painful diseases of the abdomen. The hyperesthesia of the skin, and the muscular contraction, may be general or localized, with the diffusion or localization of the internal disease.

Digestion proper begins in the stomach—a dilatation of the digestive tube, between the lower end of the esophagus and the beginning of the duodenum ; in form it is irregular, pear-shaped, somewhat flattened anteroposteriorly ; and it is lined with an exceedingly rich, glandular, internal membrane. In this viscus the action of the saliva is continued, the transformation due to the gastric secretion begins, and a second function, that of absorption, probably commences. The abnormal physical variations of the stomach are numerous and, clinically, very important. A description of its normal anatomical relations and its surface landmarks has a practical bearing.

Three-fourths of the stomach lies to the left of the median line, in the upper part of the abdominal cavity, in contact and close union with the under surface of the diaphragm. The remaining one-fourth crosses the median line in the epigastrium. The stomach lies in a nest formed by the liver, diaphragm, spleen, pancreas, suprarenal capsules, intestines, liver, and abdominal wall.

The stomach is suspended and held in position by its continuation with the esophagus and duodenum, and by its close

union with the diaphragm (gastrophrenic ligament), and with the peritoneal folds connecting it with the liver (gastrohepatic omentum), and with the spleen (gastrosplenic omentum). The greater curvature is free, and rests on the lesser omentum and on the intestines, which form for it a sort of support. The contact of the colon and the stomach may partly explain the association of their peristaltic movements. The investing peritoneum hangs in two layers from its movable lower border, and forms, with the inclosed vessels and the fat, a more or less complete covering of the intestines. A few coils of the intestines are left uncovered in the left iliac fossa, but the greater omentum may be shortened, rolled up, deformed, or imperfectly developed. It is attached to the transverse colon, and forms the gastrocolic ligament, often associating the colon and the stomach in their displacements.

The stomach moves up and down with respiration, which act serves to keep its contents in motion. Except when there are abnormal adhesions, the associated movement is not so close that the stomach can not be fixed, on expiration, by the pressure of the examining finger. This peculiarity is made use of in the differential diagnosis of the tumors of the stomach.

The direction of the long axis is from the left in front and above, to the right downward and backward, and corresponds approximately with a line slightly curved downward and joining the left nipple with the tip of the right eleventh rib. Pathologically, the acute angle formed by its axis with a transverse line across the abdomen may be increased, and with the vertical stomach becomes nearly a right angle. In the deformities of the stomach the axis is a very irregular line. In the adult, it varies in length from ten to fourteen inches, in health.

The size of the stomach is variable and is dependent on the age, and is largely influenced by the dietetic habits of the person. An arbitrary standard is valueless as a diagnostic criterion. The absolute size has no definite relation with the maintenance or loss of functional power. A change of size between two dates of examination is of value. The size can also be of diagnostic use when taken into consideration with other signs and symptoms. An inference as to the tone or the muscular power of the stomach, drawn from its size or from its capacity, is illogical and may be false. The greatest diameter, measured from the cardia, is about six inches; the greatest anteroposterior diameter about 4½ inches; and the pyloric end is about 1½

inches through. The normal capacity varies from 300 to 2000 c.c., in round numbers.

The descriptive anatomy of the stomach has a practical bearing on pathology and diagnosis. The viscus has two openings, two ends, two surfaces, two curvatures, two divisions of the cavity, and two localized, sac-like dilatations.

The two orifices are the cardiac, or esophageal, and the pyloric, or duodenal. The cardiac opening is situated on a level with the spinous process of the ninth dorsal vertebra, one inch below the diaphragm, between the lesser curvature and the beginning of the upper division of the fundus, behind the cartilage of the seventh left rib, about one inch to the left of its union with the sternum, and is covered by the left lobe of the liver. This orifice is fixed and held firmly in position by the esophagus, by the union of the peritoneal coverings of the stomach, by the diaphragm, and by the gastrophrenic ligament. It is inaccessible to palpation. The pylorus is on a level with the twelfth dorsal vertebra and the ensiform process, lower but to the right and over the lumbar attachment of the diaphragm, about two inches below the cartilage of the right seventh rib, and on a line drawn parallel to and midway between the median line of the sternum and the right border of this bone. It touches, below, the pancreas and the transverse colon ; above, the liver and the gastrohepatic omentum ; and posteriorly, the portal vein and the hepatic artery. It has no proper ligament or mesentery, is slightly movable, normally, and its displacement draws on the second part of the duodenum, which is firmly attached to the posterior abdominal wall. Pathologically, it may be found in almost any part of the abdominal cavity.

The anterior surface is directed obliquely from before and above, backward and downward, and is in relation with the left lobe of the liver, the diaphragm, and the abdominal wall. When the viscus is distended, the greater curvature rotates forward, and the plane of the anterior surface is correspondingly displaced.

The posterior surface faces backward and downward, rests upon the mesocolon, and is in relation with the third part of the duodenum, with the superior mesenteric vessels, and with the pancreas. Further behind are the aorta, the pillars of the diaphragm, and the posterior abdominal wall. The lesser curvature lies beneath the left lobe of the liver, is grasped by the gastrohepatic omentum, faces with its concavity above or posteriorly, as the stomach revolves on an axis corresponding

quite closely with a line joining the cardia with the beginning of the second part of the duodenum, and is on a level with the upper border of the first lumbar vertebra, and curves around the lobus Spigelii, the celiac trunk, and the solar plexus. Normally, it is inaccessible to palpation. On it, near the pylorus, and about four inches from the cardia, is the pyloric tubercle, the most frequent starting-point of carcinoma of the stomach.

The greater curvature begins at the cardia, curves upward beneath the left lung to the upper border of the fifth rib, and turns rapidly downward along the anterior border of the spleen. It emerges from beneath the false ribs between the cartilages of the ninth and tenth ribs, runs nearly horizontally to the median line, forming a curve which marks the location of the lesser cul-de-sac on its way across the linea alba to the pylorus. The upper part of the fundus, marked out by this line, is covered by the left lung; the second part is covered by the false ribs and by the abdominal wall. The full stomach thus extends from the fifth rib to 1½ inches above, or even below, the umbilicus.

The greater curvature approaches the lesser when the stomach is empty, and the pylorus moves to the left and lies beneath the median line of the abdomen. The empty organ lies deep within the concavity of the diaphragm, away from the abdominal wall, and is comparatively inaccessible to physical examination.

The size and form of the stomach are changed by the pressure of the surrounding organs, by the quantity and the quality of the contents, and by the tonicity and the pathological condition of its walls. In the physical examination, the relation of the viscus to the heart, the lungs, the liver, the spleen, the intestines, and the abdominal wall should be remembered.

The normal stomach is accessible to physical examination only over a limited area. This area is bounded on the right by the left lobe of the liver, above by the heart and the left lung, on the left by the spleen and the colon, below by the colon. The line of demarcation begins on the lower border of the left lobe of the liver, follows the greater curvature, or the upper border of the colon, to the cartilage of the ninth rib; proceeds upward, along the anterior border of the spleen, to the lower border of the sixth rib in the anterior axillary line, and to the right of the cartilage of the seventh rib. The area bounded by this line, and lying to the left of the left costal border, is known as the half-moon-shaped space of

Traube. Only a small triangular area of the stomach lies in contact with the muscular abdominal wall, and is bounded by the left costal border, by the greater curvature, and by the lower border of the left lobe of the liver.

The appearance of the abdomen varies with the position of the body, the age, the muscular development of the patient, and the state of the contents of the abdominal cavity. Besides, the previous contents of the abdomen leave their markings on the front of the abdomen, so that we may read on the abdominal wall, in addition to the present state, the history of gross physical changes in the contents of the abdominal cavity. The abdomen of a child—on account of the large size of the liver and the stomach, and the smallness of the pelvis and the crude muscular development—presents a different appearance from that of an adult. In a child the bladder and rectum are higher in the abdomen, and the stomach is covered to a greater extent by the left lobe of the liver.

The abdomen of the healthy and well-developed adult presents prominences and depressions corresponding to the bellies and ligaments of the abdominal muscles. The depressions are the median furrow (linea alba), between the ensiform cartilage and the lower fourth of the abdomen, and the right and left lateral furrows along the outer borders of the recti muscles. The recti muscles are inclosed in sheaths, and when strongly contracted present four prominences, marked off by transverse depressions, on a level with the eighth and ninth costal cartilages and the umbilicus. Neither these prominences nor the localized contraction of the bundles of fibers composing the muscles should be mistaken for physical changes in the contents of the abdominal cavity.

1. Inspection.—The inspection signs are remarkably true in their signification, but they are, unfortunately, also very few as regards the diseases of the stomach. But in exceptional cases inspection may rapidly reveal the nature and the severity of the disease.

In order to give unity and method to the work, we go from the general surface examination of the body to the inspection of the mouth and the throat, the general inspection of the abdomen, and, lastly, to that of the stomach itself.

The sharp and quick glance of the old clinician is a power well worthy of cultivation. It takes only a few moments to note the state of nutrition, the color of the mucous membranes, the character of the patient's expression, the condition of the skin, and the glandular swellings—which may all be connected with diseases of the stomach.

The relaxation of the muscular system may be a revealing sign. The languid, tired, expressionless face of the myoneurasthenic is well known.

In the dynamic affections of the stomach the general condition may be satisfactory, and the vivacity, the rotundity of form, and the rosy hue of health may be in vivid contrast with the nervous, restless, melancholy complaints. In anorexia nervosa and in myasthenic retention, however, emaciation may be as great as in carcinoma.

In severe diseases the adipose layer may be lost, the abdomen sunken and flabby, the viscera rolling down into the sides, away from the vertebral column, when the patient is on the back, and the skin may be peculiarly discolored, in keeping with the causative troubles. Obstruction of the cardia and pylorus, carcinoma, hypersthenic gastritis, uncompensated glandular atrophy, and hemorrhagic ulcer are the most common anatomical diseases, which may be accompanied by cachexia. The sunken eye of exhaustion and the pinched and furrowed features of the painful diseases should not escape notice. An extreme pallor of the skin and mucous membranes suddenly developing in a painful disease of the stomach, accompanied by restlessness but not by nausea and vomiting, is a valuable clinical sign of a concealed hemorrhage in an ulcer. The intensity and the association of these general inspection signs give some information concerning the severity and the possible nature of the trouble.

The inspection of the mouth, the nose, and the throat should include the state of the teeth and the tongue, and of the mucous membranes and the glands. The cause of the gastric trouble may often be found here.

The bacteria of the mouth enter the stomach through the swallowed saliva and the food. A foul tooth is a breeding nest. The teeth should be clean and the cavities filled, and losses should be replaced by artificial teeth. Even when the teeth are good, mastication and insalivation may be badly performed. The carbohydrates, when mixed with saliva, excite the secretion of the stomach more freely than when they are swallowed immediately or when they are introduced into the stomach through the tube. This result is due chiefly to the physiological action of the sugar (developed by the saliva) on secretion. The motor work is also more easily and more rapidly performed when the food is finely divided. Decayed teeth and imperfect insalivation may be the primitive and the sole causes of gastric symptoms. Correction of these defects may be all the etiological treatment that the patient requires.

The tongue is not a mirror of the stomach, as the ancients supposed. A clean tongue is an index of local cleanliness, local health, and the state of the secretions of the mouth. The coating is a layer of morbid secretion or degenerated cells, and of bacteria. The secretions of the mouth are altered, in many conditions, by the diseases of the stomach, but not in a known, a constant, and a particular manner. The tongue is a local mirror, which reflects only that which comes over its surface. A heavily coated tongue may be a cause of loss of appetite.

The character of the secretions of these parts is of more practical value. These pathological changes are excited by external or by constitutional causes. Among the many internal causes, the diseases of the stomach form one. The nose and throat changes will be described in the special section on the vicious circles of the stomach. For the present it need only be emphasized that the vicious circle is completed by the purulent, decomposing, irritating secretions from these cavities finding their way into the stomach and exciting trouble there. A chronic irritable sore throat may produce reflex nausea and vomiting. An inspection of the mouth, the nose, and the throat may reveal the source of a hemorrhage.

We begin the inspection of the abdomen after the patient has been placed in the proper position and light and the abdomen and the greater part of the chest have been laid bare.

The person lies on the back, with the spine in contact all along with the lounge, the head comfortably raised, the extremities symmetrically extended, and the whole muscular system relaxed. To hold the legs flexed on the abdomen requires effort, and makes complete muscular relaxation impossible. And what is equally objectionable is that in this position they are in the way of the examining physician. The light should fall horizontally from over the feet of the patient. In this position the patient is requested to breathe quietly and regularly, and to relax all the muscles.

The whole abdomen should be inspected, and all the deviations from the normal should be noted. But we must here limit our description to the inspection signs due to the conditions of the stomach.

It need not be expected that more than gross physical changes will be revealed by this method.

As regards the stomach, we may discover by inspection: (1) Distention of the viscus; (2) contraction; (3) displacements; (4) tumors; and (5) peristalsis.

1. The distended stomach may be clearly and visibly outlined on the abdominal wall, if the wall is relaxed and sufficiently thin. This condition may be normal after a heavy meal, but is also frequently pathological and due to retention of food and secretions, swallowed air, and, at times, to gas generated by fermentation. Grooves, marking the sweep of the greater and lesser curvatures, may be seen, and both the size and the location of the viscus may be thus truly revealed. During the period of functional repose the stomach is found distended in myasthenia with retention, and in pyloric obstruction. In the latter condition, the prominence is usually much greater than in the former, on account of the associated hypertrophy of the muscular layer. Self-inflation of the stomach by the gases of fermentation may be observed, but is of rare occurrence. The stomach fills, and then suddenly collapses as the gas escapes through the pylorus. The absence of visible distention at the moment of examination, naturally does not exclude the diseases which produce it. The form and position of the stomach are most easily seen when the stomach is full, but the significance of the distention is determined by the time after the last meal when the examination is made.

2. The contracted stomach is less easily recognizable by inspection than is the distended or displaced organ. In obstruction of the esophagus and in cancer of the cardia, the stomach may be very small and retracted. The almost empty, collapsed abdomen, with its thin covering, presents a slight prominence, which extends about as far as the lower border of the left lobe of the liver. During the paroxysms of adenohypersthenia also the abdominal wall and the stomach may both be strongly contracted; but the face displays deep lines of suffering. The stomach may be normally very small, and the accompanying emaciation may be due to diseases elsewhere than in the stomach. The stomach is often very small in scirrhus, in cirrhosis ventriculi, and in chronic starvation.

3. Inspection may give very truthful information concerning the location of the stomach. The small corset-waist, with the rounded epigastrium, suggests a deformed liver and a stomach pressed into a vertical position. The lesser cul-de-sac may then be the lowest part of the stomach, and the region just to the left of the median line and down to the umbilicus may be prominent after meals. If the stomach be prolapsed and full, the displacement may be easily recognized by inspection—the epigastrium is depressed, the abdomen

about the umbilicus—and chiefly to the left of it—is prominent, and the grooves of the greater and the lesser curvatures may be seen moving slightly with the diaphragm. If the stomach be empty, the displacement may not be noticeable, or may be less evident.

4. A visible neoplasm of the stomach is, almost without exception, carcinoma, and may often be seen moving up and down with the diaphragm. The visible neoplasms are commonly located on the lesser curvature, and are easiest seen when so located, while the stomach is empty.

It must not be imagined that a visible tumor in the epigastrium, which moves with respiration, is always a tumor of the stomach. Indeed, the visible moving prominence may not be a neoplasm. The transverse colon, when filled or distended, may often be seen moving up and down as a prominence on the thin abdominal wall, in the lower third of the line joining the ensiform process and the navel, and to the left or the right of the median line, or both. This ridge is often mistaken for the lesser curvature, and a gastroptosis is supposed to exist; or it is mistaken for a gastric neoplasm. The respiratory movement of the abdominal organs is not dependent solely on an attachment to the diaphragm, but all of them move which are caught in the swing between the diaphragm and the abdominal wall, or are influenced by the increase or the decrease of intra-abdominal pressure. The closer, however, the union is with the diaphragm, the greater is the difficulty of fixation on expiration.

5. The peristaltic movements of the displaced or obstructed stomach are often visible. These peristaltic waves come out from beneath the left false ribs and roll across the abdomen to the pylorus. When most violent, the peristalsis may be followed by a visible antiperistalsis. The peristalsis may be excited when the organ is moderately filled with cold water, by ether sprayed over the epigastrium, by faradization, by an effervescent drink, or, more conveniently, by a few gentle strokes with the tips of the fingers. Visible peristalsis results from the strong efforts of the stomach to overcome an obstruction to its evacuation. It is most common in pyloric or duodenal obstruction, in gastroptosis with angular constriction of the duodenum, and it exists rarely as a particular dynamic affection of the stomach. In cancerous obstruction of the pylorus it is much rarer than in benign obstruction with excessive secretion.

The loss of tone of the abdominal muscles plays an important part in the pathology of the stomach. In health, the

elasticity of these muscles subjects the abdominal contents to a limited degree of pressure, and affords them a certain amount of support conducive to their nutrition and to the performance of their functions. Abdominal tension also regulates the lymph and venous circulations of the abdomen. The flabby wall exerts no pressure, and the weight of the unsupported stomach drags on its attachments. Inspection may at once reveal this half-filled bag-like abdomen, which is an inspection sign, not properly of the stomach itself, but is often associated with the displacement of this viscus and with a poor abdominal circulation. The inspection of the abdomen yields both positive and negative information. Some diseases of the stomach display no visible physical changes; some are made manifest by particular inspection signs. A negative result of inspection excludes with probability the latter class of diseases, and limits investigation to those diseases which yield no inspection signs at any period during their evolution.

The stomach may also be inspected during its illumination. For this purpose the stomach-lamp and the gastroscope may be used.

2. Electric Illumination of the Stomach.—A simple and practical method of illuminating the stomach was first devised and employed for the purpose of diagnosis by Einhorn. Other cavities of the body had already been successfully illuminated, and Milliot had employed the electric light to illuminate the stomach of the cadaver and of animals. But for a simple method of illuminating the human stomach during life for the purposes of diagnosis, we are indebted to the inventive genius of Einhorn. The instrument consists of an ordinary stomach-tube with a small Edison lamp, which is inclosed in a thick glass capsule at the lower extremity (Fig. 1). The conducting wires run in the lumen of the tube and connect with a storage battery. Some of the instruments now in use have openings for the introduction and withdrawal or continuous circulation of water. The lamp can be kept cool without the use of a continuous current, and cleanliness demands the use of a tube without openings. The electric illumination of the stomach has been thoroughly studied by Einhorn, by Heryng and Reichman, by Kuttner and Jacobson, and by Meltzing, and others. Some of these writers claim for this method of examination greater accuracy and utility than, in our opinion and experience, it possesses.

Before the examination, which should be made in a dark room, the stomach is thoroughly washed out, unless it be

already empty. Two or three glasses of water are administered, or introduced through the tube before it is withdrawn. The electric lamp is next introduced in the same manner as the ordinary stomach-tube. The patient usually stands during the examination, but may also lie on the back. The current is turned on and broken at short intervals in order to avoid heating the lamp. When the light is turned on, the illuminated stomach is displayed as a bright area on the abdominal wall, shaded by the recti muscles, crossed by the veins, and



Fig. 1.—Ewald's Einhorn stomach-lamp.

bounded in part and at times by the curvatures of the stomach. After the general illumination, the lamp may be pushed further on toward the pylorus and made to wander along and to locate the greater curvature as the tube is slowly withdrawn. The water may be left in the stomach.

The information obtainable by the electric illumination of the stomach is of both positive and negative value. By it some of the diseases of the stomach can be excluded, while others, particularly gastroptosis, may be revealed by it. But

the utility of the method is limited in practice, and the information obtained by it is very liable to be misinterpreted. It is only practicable when the patient quietly tolerates the presence of the tube. It naturally excites some fear, and many patients accustomed to the use of the stomach-tube reluctantly consent to the employment of the light. There is very little, indeed, of the information obtained by it that is not given with greater accuracy by other and simpler methods. That the instruments are expensive and the method of using them is but little understood, can not be considered valid objections. At present, however, electric illumination is properly used to confirm or control our conclusions or suspicions, and is most likely to be valuable in obscure cases where it is advisable to avail ourselves of every possible source of information. It may be employed to determine the position and size of the stomach, the location of the pylorus, and the existence, origin, and location of tumors.

The illuminated area visible on the abdominal wall does not always represent the size or form of the stomach. The left lobe of the liver shuts off a part of the light, and, when enlarged, may be misleading. The transverse colon, when filled with gas, may also be illuminated. The general illumination of the stomach possesses a negative value when the bright area corresponds with the size and position of the normal stomach. In gastroptosis and in vertical displacement of the stomach, the form and position of the illuminated area may be so clear and characteristic as to admit of no doubt. The result can be confirmed by allowing the lamp to wander with intermittent flashes, as recommended by Meltzing.

The electric lamp may also be used to locate the pylorus, the point where the wandering light stops or turns its course backward along the lesser curvature being noted.

It is exceedingly rare that a tumor of the stomach originates on or extends to and involves the anterior wall, without being revealed by inspection, palpation, and inflation. But the electric light may be used to confirm a diagnosis of a tumor of the anterior wall, or to prove that the anterior wall is not thickened by a neoplasm or by infiltration. It should not be forgotten that fecal masses may get between the stomach and anterior abdominal wall, or that new growths originating in other parts may obstruct the light, or that a localized dark spot may have its cause in the abdominal wall itself. Dark spots due to fecal masses are not constantly

present, and may, by manipulation, be removed from their position in front of the stomach. We have had no experience with Röntgen's rays.

The Gastroscope.—By means of the gastroscope constructed by Leiter, the interior of the stomach may be inspected. Mikulicz studied clinically a number of cases of carcinoma and of ulcer with this instrument. The use of the costly and complicated metallic instrument is a severe operation. Rosenheim has recently modified the gastroscope, but has not succeeded in increasing its utility in private practice.

3. Palpation.—The value of the information obtained by palpation is conditioned by the proper performance of the work by fingers which have been educated for this special purpose. Each palpable organ, in health and disease, imparts to the hand particular sensations, which enable us to separate one organ from another, and to detect changes in form, in size, in location, in sensitiveness, and sometimes in texture. By palpation of the stomach and intestines it is also possible to obtain some information concerning the quantity and the physical qualities of their contents.

The general palpation of the abdomen should be made with the hand open, all hand and arm muscles being in repose. In this natural and easy position of the hand the fingers are slightly flexed, and the sense of touch is most delicate. The warm hand is next placed on the abdomen, preferably with the fingers lying crosswise in relation to the axis of the body so as to appreciate the respiratory movements of the abdominal organs. The pressure should be light at first, and then somewhat firmer—the hand remaining still while the patient breathes naturally without effort. The hand may next be slightly moved back and forth over the different regions of the abdomen and in a direction at a right angle to the long axis of the part of the digestive tube contained therein. In the palpation of the particular organs or of their parts the palmar surface of the fingers is used. Both hands should be educated to do the palpation, but one must frequently be employed to bring a part within reach, to fix it, or to relax the abdominal wall. Gentleness, ease, method, and the use of the most sensitive part of the hand—these are the requisites of palpation. A brutal thrusting of the tips of the fingers would cause pain, would excite contraction of the abdominal muscles, and would, possibly, produce injury; while hard pressure would destroy all delicacy of touch.

The conditions under which palpation is done also modify the results. The intestines, particularly the transverse colon,

should always be empty, when the exploration is concerned with the stomach alone. The lowered abdominal tension produced by evacuation of the digestive tube and the bladder facilitates the examination of the abdomen; but the artificial evacuation of the stomach and intestines may not aid in the discovery of their pathological states, and the search for the cause of a digestive trouble is never complete until after the careful palpation of both these divisions of the digestive tube. Consequently, the first examination should be made without preparation, the patient not being under the immediate influence of or recovering from the effects of drugs. After the first examination the bowels, if necessary, should be thoroughly evacuated. To acquire all the information possible by palpation of the stomach, the exploration should be made under low abdominal tension and complete relaxation of the abdominal muscles—while the organ is digesting, while it is empty, and after inflation. Rarely, it may be necessary to conduct the examination under anesthesia.

The physician should be comfortably and securely seated at the right of the patient, facing the patient's head, and with the arms and the warm hands free. The usual position of the patient is that occupied during inspection—straight, relaxed, flat on the back. Other special positions may be advantageous in particular cases. A very useful position for palpating the pylorus and the part of the stomach lying beneath the left costal border, is on the left and right side respectively, with the chest slightly elevated and with or without the legs flexed on the abdomen. The knee-chest position and the erect position (when the lower part of the abdomen should be supported) may also be employed in special cases. If the patient be placed on the back, with the chest raised and supported, and with the legs strongly flexed on the abdomen and supported in this position, the deep palpation of the abdomen is wonderfully facilitated. Whatever position the patient assumes during the examination, his abdominal muscles must be inactive and relaxed.

The palpation of the abdomen should proceed methodically, by regions and by organs, and the resistance, the tenderness, and the physical abnormalities of each area and each organ should be noted. After the general palpation of the abdomen, each important abdominal organ should be examined. At the bedside we adopt the following order: the large bowel, the kidneys, the liver, the spleen, and, finally, the stomach. The position and the excursion area of the diaphragm should be determined by observing its line of movement on the chest-

wall, and by percussion and auscultation of the heart and lungs. We thus begin the examination of the stomach with a great deal of important information concerning the location and physical properties of the organs around it.

The palpation of the stomach will be discussed under the following divisions: (1) Palpation during digestion, during the period of repose, and after inflation; (2) Palpation of the tumors of the stomach; (3) Tenderness.

1. The stomach, on palpation, may not be found empty during the period of normal physiological rest. This condition is always pathological, and is due either to myasthenia or to obstruction, or to excessive secretion.

The palpation of the stomach during digestion may afford some information concerning its position and the state of its muscle. The hand, gently placed over the digesting organ, may often feel it alternately contracting and relaxing, in the performance of its churning and evacuating functions. Persistent contraction is a sign of continuous muscular excitation. The resistance to the hand may be continuous, and associated with stormy peristaltic efforts in obstruction. The myasthenic organ may be found flaccid and non-resistant.

Palpation of the moderately full or the distended stomach may also locate its position, and may reveal a displacement. Two methods of palpation are useful in locating the position of the stomach: The organ and its lower limit, or both its upper and lower limits, may be felt by the full hand moved from the most prominent part of the stomach toward its upper and lower limits. Sometimes the stomach can be felt moving up and down beneath the hand during respiration. The second method is often very exact, and is useful both when the stomach is empty and when it is moderately full. To locate the lower border, the palmar surfaces of the fingers of both hands are placed on the abdomen, and lie in the direction of the long axis of the body and on either side of the linea alba. The patient is told to breathe deeply and regularly. Near the end of inspiration the fingers are gently and firmly pressed downward so as to compress whatever lies between them and the vertebral column. During expiration the depressed fingers are all simultaneously drawn down toward the symphysis. The procedure is repeated, now higher and now lower, until the point where the lower edge of the stomach is felt to slip up beneath them during expiration is reached. The same method may be employed while the diaphragm is at rest. To locate the upper border when it is made accessible to palpation by gastroptosis,

the fingers should point toward the symphysis and be drawn toward the ensiform process while the abdomen is in repose. The edge may thus be felt to slip suddenly from beneath the fingers; or the fingers may be placed as in the method of locating the lower border, and gently but rapidly drawn from their point of pressure on the spinal column toward the symphysis pubis, during expiration. The ridge of the lesser curvature may sometimes be detected in this manner. When the stomach is empty, the examination is facilitated by the administration of an effervescent drink. The differentiation of the stomach from the pancreas and from the transverse colon presents no difficulty to one conversant with anatomy and experienced in abdominal palpation. The pancreas, if accidentally felt (apart from its location and its small width and thickness), can be easily differentiated by its immobility and absolute stillness during respiration. Whenever one border of the transverse colon can be felt, the other border can be felt also, and the colon is nearly always narrower than the stomach. In case of doubt, the other methods of locating the lower border of the stomach and of differentiating the colon from the stomach can be used. One of the surest is the palpation of the tube when introduced into the stomach.

Leube introduced a method of locating the lower border of the stomach which, however, he now no longer employs. A stiff sound is introduced into the stomach, and pushed on until the resistance of the lower curvature is felt. By palpation through the abdominal wall the lower end of the sound is located. Meltzing allows the electric light to wander along the curvatures of the stomach, and its visible line of march is thus located. Turck uses a revolving sponge, which is palpable and sometimes visible along the line of its march in the same manner. Boas recommends the employment of the stomach-tube, the position of which is located by palpation as it glides and lines itself along the greater curvature. These methods, on account of the distensibility and easy displacement of the borders of the stomach by pressure, may give too large an area, but are sufficiently accurate for all practical purposes, and, when the tube is tolerated, are most excellent control methods. The spiral stilet pyloric sound of Kuhn (Fig. 2) is better than the simple stomach-tube; and both of these instruments, when they are employed for locating the greater curvature, the pylorus, and the tumors of the stomach by palpation, and for differentiating the tumors of the stomach from the tumors of adjacent organs, should be about

100 cm. long. It is rarely necessary to introduce more than 75 cm. of the tube; and the examination should be made when the stomach is empty and when it is full, when the



A B

Fig. 2.—A, Kuhn's pyloric sound. The spiral metallic stilet (*a*) is to be introduced into the stomach-tube (*b*), made of Jacques' rubber; B, Kuhn's balloon sound. The stilet (*x*) fits into the rubber tube and inflatable bulb (*y*).

patient is lying flat on his back and when in the erect position.

2. Not all the palpable tumors of the stomach are malignant. They may be due also to ulcer, with adhesions and exudation,

to simple hypertrophy of the pylorus, and, very rarely, to benign neoplasms. A very valuable differential sign of a gastric tumor is that it may be fixed by the palpating fingers during expiration. But when adhesions have been formed to the diaphragm, or to the organs closely bound to it, this differential characteristic is lost. The gastric tumors, like those of all organs attached to the diaphragm, move up and down with this muscle, in respiration. The tumors of the stomach always feel much smaller than they are, and, though fixable on expiration, readily slip up when the pressure of the fingers is lessened.

A negative result of palpation in a case of suspected gastric tumor means very little. The tumors of the cardia can not be felt. The same is true when they are small, wherever situated, and when the conditions are not favorable to a thorough examination. Cancer may be diffused, and may thus escape detection. Tumors situated posteriorly are never palpable, unless the viscus be empty. The tumors of the lesser curvature are turned back, out of reach, when the stomach is distended, and may be drawn up beneath the bony thorax by adhesions to the diaphragm. The tumors of the pylorus may just as often be felt elsewhere as at the point marking the situation of the normal pylorus. These tumors may move only with the diaphragm, or may, rarely, be carried by the fingers into almost any part of the abdomen.

It is often difficult to decide whether the tumor belongs to the pylorus. Inflation of the stomach may clear up the difficulty. A better and simpler way is to give the patient a glass of water, and to hold the fingers gently on the pylorus, when the part will grow alternately soft and resistant, except when the pylorus is converted into a hard ring by an infiltrating scirrhus, and the water can be felt bubbling through. A spurting sound, somewhat like that at the cardia on swallowing, may also be heard with the stethoscope, and is loudest directly over the pylorus. Or the pylorus may be located by Kuhn's pyloric sound.

The palpation and localization of the tumors of the stomach may be wonderfully facilitated by the employment of Kuhn's or Schreiber's balloon sounds, which are useful, also, for locating the stomach and the cardia, and for detecting the bilocular stomach and the incomplete division of the stomach into two cavities by deformities or by compression. The little balloon is inflated to a suitable size after the passage of the cardia or after the passage of the constriction of the body of the stomach, and the cardia or the constriction

is detected and located in the manner employed for the detection and location of a urethral stricture.

3. Epigastric tenderness may or may not be due to gastric disease.

It is very common to find the abdominal muscles sore after straining or unwonted use of them. The soreness corresponds with the area of the muscle, and not with the distribution of the cutaneous nerves.

Increased sensibility of the skin over the epigastrium, and corresponding with the distribution of the cutaneous nerves, reflects a morbid sensibility of the gastric mucous membrane, and is a very valuable palpation sign. The route of the reflected hyperesthesia passes through the vagosympathetic ganglia and the cord.

Epigastric tenderness is frequently due to the sensitive left lobe of the liver, the whole of which, or only its border, may be painful on pressure. The tender area or line will be found to correspond with the size and the form of the left lobe, and will at once suggest its cause. The other lobes of the liver may be tender; the area of the stomach itself is not so. Epigastric tenderness may be due to an irritable solar plexus. The pain on pressure is somewhat dull, unnerving, and considerably affects the circulation. The tenderness of the plexus is greatest during digestion, and may disappear during the period of repose.

The tenderness may be in the duodenum. This is the case in duodenitis, duodenal ulcer, and duodenal stagnation. The point is near the middle of the right costal border, and gentle but firm pressure should be exerted upward and outward and backward. Phillip located this tender point in the pylorus. Glénard places it in the quadrate lobe of the liver. It may also be in the head of the pancreas or in the choleduct. In gastroptosis, under favorable circumstances, the pancreas may be felt as a flat, thin, immobile, transverse body, which should not be confounded with the transverse third part of the duodenum, which is lower. Pressure on the pancreas, when thus found, is often painful. Here the tender point of Phillip may also have its seat.

The colon passes along the lower border of the stomach, and the pain on pressure located therein should not be confounded with that of the stomach.

Diffuse gastric tenderness may be muscular, as after gastric spasm, or may be due to anatomical disease of the lining membrane, to cancerous infiltration, to perigastritis, and, possibly, to gastralgia. This diffuse gastric tenderness possesses a very

indefinite meaning. This is not true of the very sharply limited, small, often exceedingly tender spot located on or near the median line, below the xiphoid process, and associated with a circumscribed tender spot to the left of the twelfth dorsal vertebra. These palpation signs are most common in ulcer, but they may also be due to tender adhesions, and, rarely, to carcinoma. The ulcer pain may be excited by a pressure of one or two pounds; that of the other painful diseases of the mucous membrane of the stomach requires from three to ten pounds. The algesimeter of Boas is a very useful instrument for measuring this difference with exactness.

Gastric carcinoma may also be exquisitely painful on pressure, and this quality may aid us in differentiating it from painless tumors, true or false.

Palpation, like inspection, yields both positive and negative information, which may reveal a disease or a group of diseases of the stomach.

4. Percussion.—The area of the stomach which can be marked out by percussion is dependent not only on the size, the position, and the contents of the stomach, but also on changes in the surrounding organs, the liver, the lungs, the heart, the pleura, the spleen, and the colon. Large abdominal tumors would alter its percussion boundaries. Consequently, the abnormal percussion signs should be used with circumspection in implicating the stomach as the diseased part.

In percussing the stomach, we locate successively the upper, lower, right, and left borders, and note the occurrence of pathological sounds within this area. The percussion limitation of the stomach should be attempted only when the stomach is moderately full of gas. If the stomach be empty and normal, it is withdrawn into the concavity of the diaphragm, and the transverse and splenic flexure of the colon occupies the vacated spot in the epigastrium. If distended, the stomach limits will be too high or too low.

The location of the upper border is found by moderately deep percussion from above downward. According as the percussion is deep or shallow will the result be slightly different, and the transition from pulmonary resonance and from dulness to gastric tympanicity will be more or less abrupt.

If the organs of the thorax are healthy and the stomach is moderately distended with gas, the normal superior percussion limit is approximately the following: In the left parasternal line, behind the fifth rib; in the clavicular line, in the fifth intercostal space; in the anterior axillary line, which is

the furthest extension of the stomach to the left, in the seventh interspace.

The slight displacements of the upper border are of little value in the diagnosis of the diseases of the stomach. The only diseases of the stomach which modify greatly the position of the upper border are gastroptosis, upward displacement, and left subphrenic abscess complicating an ulcer or a malignant growth. In gastroptosis, the lesser curvature descends simultaneously with the fundus and with the greater curvature. The limit may be lowered by left pleurisy with effusion, pneumonia, emphysema, or pneumonia of the left lower lobe. It may be raised by conditions causing ascent of the left cavity of the diaphragm. The upper percussion border is also modified by changes in the size of the left lobe of the liver, and by the gaseous distention of the stomach.

The location of the lower limit of the stomach by percussion is more difficult than the location of the upper boundary. We begin with very light percussion (the patient lying on the back, as usual) in the prolongation of the left parasternal line near the symphysis, and gradually move upward, noting, if possible, the transition from the sound over the colon to that over the stomach. There is often a narrow transition area over which the two sounds are mixed, and may be separated and different notes produced by closing or extending the gently percussing fingers at the moment of contact with the one resting on the abdominal wall. The supposed gastric sound should then be followed to the left and right, to see if it corresponds with the form and the location of the stomach or the colon.

Special devices have been found useful and necessary in the delimitation of the lower border by percussion. One consists of the introduction of fluid or gas into the stomach or colon, so as to produce a corresponding difference in the percussion note. If, for example, the stomach and the colon give a clear and a similar tympanitic sound, we first note this fact in the erect and in the recumbent position. We next give the patient a glass of water, and mark in the erect position the area or line of diminished clearness, which again becomes clear in the recumbent position. Or a quart of water may be introduced into the stomach through the tube, the area of dulness produced thereby located, and, in order to prove that the dulness is due to the contents of the stomach, the water is again removed by expression (Piorry, Penzoldt). Some notion of the tonicity of the stomach in myasthenia may be gained by giving the water in successive half-glass-

fuls, and by noting the descent of the line of diminished clearness (Dehio, Boas). Whatever devices may be employed, the percussion signs are not so markedly changed as we should anticipate, and often leave us in doubt. The descent of the normal lower boundary means that the stomach is enlarged, distended, myasthenic, or displaced. Two or more of the conditions may be combined in a particular case, and the differentiation must be made by other methods.

The right limit of the percussion area of the stomach may be most easily marked out by proceeding with light percussion from over the tympanitic stomach, along parallel lines, to the right. The points of beginning dulness mark the right border accessible to percussion. The position of this border varies with the size of the liver.

If the liver be normal in size or small, a small area of gastric resonance may be located across the median line, below the left lobe. In myasthenia, or in enlargement from obstruction, the lesser cul-de-sac may extend downward, and further to the right. In vertical displacement, the percussion area does not cross the median line.

The left percussion border is displaced chiefly by enlargement of the spleen.

The changes in the percussion note over the normally tympanitic area of the stomach, uncovered or unoccupied by a healthy or a diseased adjacent organ, may be due to the contents of the stomach, or to a tumor.

It is quite characteristic of the moderately filled myasthenic stomach that the percussion sound should vary greatly, both with the position of the patient, and with the peristalsis of the organ. The well-toned normal muscle contracts on its contents, and maintains a notable degree of intragastric pressure. The normal tonicity tends to maintain the form, despite changes of position of the body. The flabby, myasthenic stomach alters its form, in obedience to the laws of gravity, and to the slight pressure of neighboring parts. These variations may be noted during digestion, or during the period when the stomach should be empty.

During digestion there is more or less dulness over the fundus, from one to five hours after a meal, in accordance with the quantity and quality of the food taken. In the strong stomach, the left or upper boundaries move to the right or downward, as the patient rolls to the right side or stands erect. Under the same circumstances, the fundus of the myasthenic stomach becomes tympanitic, but in the erect position the lower boundary sinks notably.

The percussion note may vary without change of the position of the patient. A point now dull becomes, after a few minutes, tympanitic, and vice versâ. The phenomenon is due to peristalsis, usually in a flabby, myasthenic stomach partly filled with fluid and gas.

Before dismissing the subject of percussion, we wish to make an emphatic protest against the too great value ascribed to the mere size and to the percussion limits of the stomach. Much of the time spent in the exact location of its borders is wasted. The capacity of the stomach bears no relation whatever to its functional efficiency. The location of the borders may enable us to say whether the stomach is larger or smaller than the average; but we can not say whether the size is or is not normal for the individual under examination. The limits of the stomach are changed by a few of the pathological conditions of the stomach itself, such as displacement, obstruction, distention, stenosis of the cardia, and, sometimes, myasthenia. Concerning these, percussion may only give vague suggestions that must be controlled and complemented; but often the limits of displaced organs may be located with precision, as when the lesser curvature, on account of gastroptosis, emerges from beneath the left lobe of the liver, or when the wandering pylorus is dragged down by a neoplasm.

The percussion signs when negative are valueless, in this respect differing from those obtained by inspection and palpation. When positive, they are inaccurate. The lower border, being distant from the abdominal wall, is always lower than percussion places it. The right and left borders can not be even approximately determined. The contraction and thickness of the abdominal wall modify the percussion note. These signs are approximately accurate only when the stomach is just full of gas without distention. Of more importance than the mere position of the borders are the difference in their location when the stomach is empty and when it is full, and the variations in the distance separating the upper and the lower borders measured on the parasternal line.

The distance from the cardia to the greater curvature may be measured by the method of Purjesz. The external end of the stomach-tube is connected with a manometer. The passage of the tube through the cardia and its entrance into the stomach are marked by a sudden change from negative to positive pressure. At this moment a mark is made on the tube where it crosses the incisor teeth, and it is then pushed

on until the resistance of the greater curvature is felt. The length of the tube required to extend from the cardia to the greater curvature measures the distance between them.

More accurate than simple percussion is auscultatory percussion, which may be performed in two ways: The binaural stethoscope may be fixed over the triangular space where the full stomach comes in contact with the abdominal wall, and percussion may be performed along eccentric lines running in every direction until the sound is lost; or the stethoscope may be moved along these lines while the percussion is being performed over the triangular space. Combined percussion and auscultation are not likely to be employed except in obscure cases, when percussion signs are more likely to deceive than to instruct.

5. Inflation.—Inflation of the stomach is a device which may render much more exact the results of inspection, of palpation, and of percussion.

The older method consists in the administration, successively, of tartaric acid and of bicarbonate of soda, the CO_2 set free in the stomach distending the organ. Some use as much as one dram of the acid, and a little more of the bicarbonate of soda. These large doses may produce discomfort, but may be required to fill stomachs of more than average capacity. It is seldom advisable to use more than one-half a dram of tartaric acid, dissolved in one-third of a glass of sweetened water, and 35 grs. of the bicarbonate, also dissolved in a small quantity of water, when the object is to determine the boundaries of the stomach by percussion. But to render the stomach visible and easily palpable, the large doses of the tartaric acid and soda are necessary. The patient drinks the acid solution, waits about one-half of a minute until it has all been emptied into the stomach, and then swallows the solution of the bicarbonate of soda, closes the mouth, lies flat on the back, and breathes quietly. The acid requires about one-ninth more of the bicarbonate of soda for saturation.

The second method consists in the introduction of air into the stomach through the tube. Bouveret suggests that the physician apply his mouth to the end of the tube and thus inflate the stomach, but this method of inflation is objectionable on grounds of cleanliness, and the physician's mouth is in danger of becoming filled with regurgitated stomach-contents. The tube being introduced, the bellows part of a double-bulb atomizer is attached to it by means of a piece of glass tubing, and the stomach of the patient (who lies on

the back) is then very slowly and watchfully distended with air. The inflation should be at once stopped if the patient show signs of distress, even though the stomach has not been well distended.

Each method has its advantages and disadvantages. The inflation with air is under the control of the operator, and the air can be increased or diminished and the operation suspended or repeated at will. But the method is greatly limited by the necessity of employing the tube, and the operation should never be attempted before the patient has lost all fear

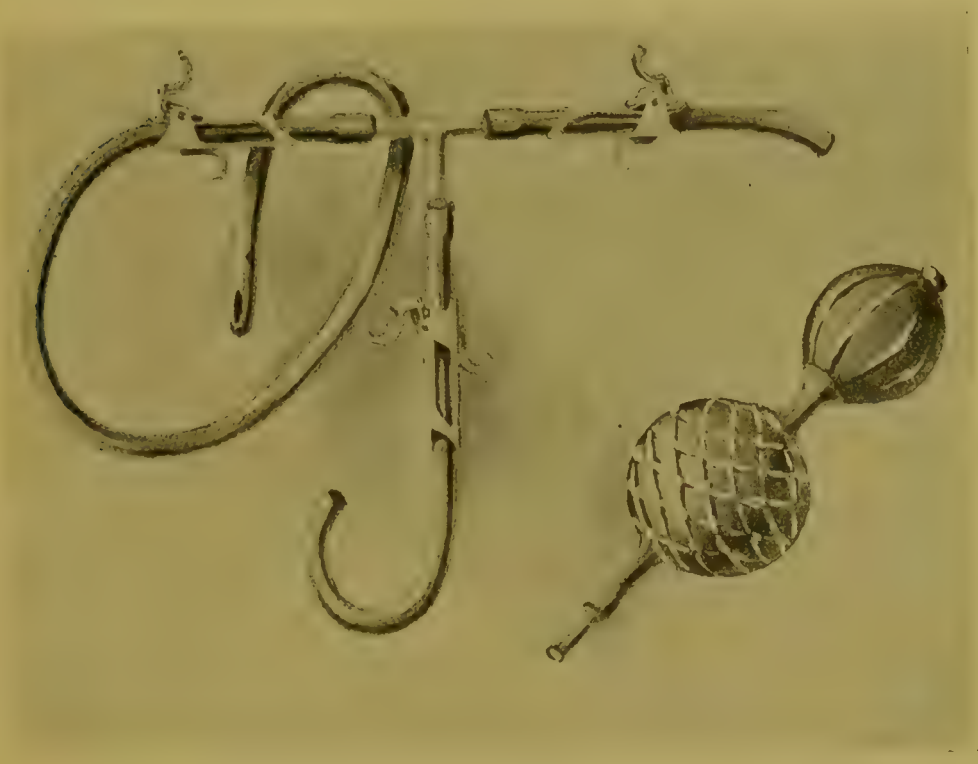


Fig. 3.—Strauss' apparatus for lavage and inflation.

and has learned by experience to tolerate the tube. The distention with generated gas is more universally applicable, but is uncontrollable, and may be unsatisfactory. Both methods may be rendered useless by the rapid evacuation or passage of the gas or air into the duodenum. When the pylorus is incontinent, the gas bubbles through it rapidly and continuously. The cause of the failure of the pylorus to close properly (atony, paresis, infiltration, particularly by cancer, and peripyloric adhesions) may not be revealed by this method and may be left an open question to be answered by

other signs and by the clinical history. More commonly the gas is rapidly evacuated by peristalsis, when its intermittent passage through the pylorus may be felt and heard.

Inflation should not be used, unless valuable information in the particular case is likely to be gained by it. But a more important contraindication is furnished by the liability to do injury. It should not be employed in ulcer nor in advanced carcinoma, nor where the clinical history makes it likely that perigastritis and peritoneal adhesions exist. The carbon-dioxid method should not be used when flatulency produces great distress or gastric spasm, or when there is gastric retention. The other contraindications are those against the use of the tube. It is unnecessary to say that neither method should be employed during the period of physiological activity of the stomach. If the acid and soda method of inflation be selected, it is a good plan to have a stomach-tube ready for the removal of the gas whenever (as almost never happens) the gaseous distention produces much discomfort.

Inflation gives the best results with thin abdominal walls, when the limits of the stomach may be plainly seen, felt, and marked out by percussion. It may reveal, as no other method does, vertical displacement, total descent, and enlargement of the stomach ; or it may exclude these conditions. The tumors of the lesser curvature may be turned back out of reach, those of the pylorus may be revealed, while those of the greater curvature and of the anterior wall may become less distinct and less sharply limited. The tumors of the posterior wall are further removed from physical examination.

The inflation is useful not only in determining the location, size, and form of the stomach, but also the presence of adhesions and the size and origin of tumors. To reveal the capacity of the stomach and to determine the strength of its muscular layer the method is worthless clinically, even though the quantity of air used be measured, inasmuch as part of it may escape through the pylorus or cardia ; and the reflex muscular activity and the degree and ease of distention do not measure the retraction power of the stomach. Schreiber, Jaworski, Kelling, and Ost have described methods for estimating the capacity of the stomach. The principle embodied in all these methods is the same, but different means are employed for measuring the quantity of air that can be introduced into the stomach before the distention becomes painful. These methods are not sufficiently accurate nor simple to be of much clinical value. The information given by any method concerning capacity is of little value.

6. Auscultation.—Gastric sounds have not been studied with the care that the subject deserves. The information obtained by the sounds produced at the orifices and in the body of the stomach may aid in the discovery of abnormal conditions. These auscultation signs are the deglutition sounds heard at the cardia; the pyloric and perforation evacuation sounds; the peristaltic, cardiac, aortic, respiratory, and fermentation sounds; succussion and percussion splashing.

The deglutition sounds may be changed in the diseases of the cardia, but not, however, by diseases of the cardia only. The esophagus also plays an important part in their production. But apart from diseases of the esophagus, the deglutition sounds may be given a negative diagnostic value in that their presence and their normal qualities exclude certain disorders of the cardia.

The deglutition noises are two in number: The first follows immediately after the patient swallows the mouthful of water, and is forcible, spurting, quick; the second follows in from five to fifteen seconds, and is dull, labored, and bubbling. When both these signs are normal we may give them a negative meaning, with great probability, and may exclude organic and spasmodic obstruction of the cardia. Both deglutition sounds, particularly the first, may be absent at times in health, but their constant absence is evidence of the complete, or almost complete, obstruction of the esophagus or of the cardia. In spasm of the cardia both sounds are often delayed. The delay and prolongation of the second sound is a sign of stenosis of the cardiac orifice, the two sounds being often separated by an interval of about one minute. The sounds are best heard to the left of the ensiform process, in front; or on a level with and to the left of the spinous process of the ninth dorsal vertebra, behind. It seems, after careful study, more than probable that the two sounds mark the noisy beginning and ending of the same process, the interval between them being occupied by the entrance of the water into the stomach. The test should be made with a full swallow of water.

The pyloric evacuation sound is of a quick, spurting, bubbling, metallic character. This auscultation sign may become very important in special conditions, and is of very much greater diagnostic value than the cardiac deglutition sounds. The pylorus may be auscultated during digestion, or when the stomach should be empty, or after the patient has taken a glass of water. During the period of repose of the normal

stomach no sound is heard in the pylorus. If a glass of water be given, the sound, recurring at intervals of about one minute, may be very plainly heard with the stethoscope placed over the pylorus. The pyloric evacuation sound may be utilized to locate the pylorus when it can not be felt, and also to identify it when displaced. It is also a measure of peristaltic activity. In myasthenia, the beginning of peristalsis after a meal is delayed, the peristaltic intervals are long, and the pyloric evacuation sound may be heard when the stomach, if normal, would be empty. In compensated obstruction, the peristalsis is often quick and powerful, and accompanied by a regurgitation sound, which is different in character from the firm pyloric spurt. Though peristalsis be active, the sound is absent during pyloric spasm. It is never heard in complete obstruction of the pylorus.

In perforation of the stomach, cases have been reported in which the intermittent escape of the contents into the peritoneal cavity could be heard. This auscultation sign is rarely sought, and has not been so clearly defined as to be employed in diagnosis.

The fermentation sounds in the stomach may often be heard in stagnation and retention, with active formation of gas. They are very fine crackles, like the bursting of numerous bubbles in a partly filled vessel, and can be artificially produced by the administration, separately, of a little tartaric acid and soda. They may be most easily found if, after the patient has remained perfectly motionless on the back, breathing quietly and regularly for a few minutes, the stomach be slightly agitated while the stethoscope is placed over it. If these sounds are heard during the period when the stomach should be empty, they denote that the stagnation or retention, as the case may be, is accompanied by gas-forming fermentation.

Sounds are also produced in the stomach by peristalsis. These gentle, flowing, rushing, bubbling sounds occur normally during the period of digestion, and are usually loudest near the pylorus. If heard during the period when the stomach should be empty, they denote either stagnation or retention.

In abnormal conditions, the heart sounds may be heard over the region of the stomach, and may possess metallic, resonant qualities. This physical sign may be due to distention of the stomach with gas, to upward displacement of the stomach, to pericardiac adhesions, or to subdiaphragmatic abscess. It denotes an abnormal condition of the heart, or

of the stomach, or of their relations; and when it is constant, a search for the significance should be made.

The respiratory murmur of forced breathing may sometimes be heard over the stomach in perigastritis, in pleurodiaphragmatic adhesions, in peritonitis, and in ascites. The pulsations of the aorta may rarely produce intragastric sounds, and an aortic bruit may be transmitted and its qualities modified by the contents of the stomach. These auscultation signs are of no value in the diagnosis of the diseases of the stomach.

Intragastric noises are very frequent and important auscultation signs. For diagnostic purposes, they may be separated into gurgling, clapping, and splashing, the other intragastric sounds having been already described.

Gastric gurgling may be respiratory, or may be elicited by the physician alternately compressing two compartments of the stomach, or by the gliding method of Glénard. Its production requires the presence of very special conditions—a flabby stomach containing gas, or gas and liquid, separated by a constriction or by compression into two cavities, the contents of one of which are forced into the other. A certain relation must exist between the properties of the contents of the compressed cavity, the size of the communicating canal, and the tension of the receiving cavity, otherwise gastric gurgling can not occur.

Respiratory gurgling is more frequent in women than in men. The corset or the belt, the left arm laid across the abdomen while the person lies on the back, the colon, the enlarged spleen, the enlarged left lobe of the liver, a tumor, adhesions, or constricting bands deforming the stomach and dividing it into two pouches, may induce constriction or compression. In the majority of cases the stomach is displaced and myasthenic. The compression of the cavity from which the contents are driven is made directly by the descending diaphragm, or indirectly by the ascent of the diaphragm. A double respiratory gurgle is not rare. The respiratory gurgle is pathological, and indicates myasthenia, displacement, deformity, or abnormal compression. If it occurs at a period when the stomach should be empty, it reveals stagnation or retention; for the non-functionating stomach should retract and contain no fluid and little or no gas.

Under suitable conditions, gastric gurgling may be produced by the gliding method of Glénard. The stomach is gently compressed against the vertebral column by the

border of the hand transversely placed across the abdomen—at the end of inspiration—in such a manner as to shut off a pouch from the general cavity of the stomach. The line of compression is moved downward during expiration, and the gurgle is produced. Glénard regards this as one of the signs of the descent of the pylorus, and claims that the intragastric pressure is lower than normal, and that the stomach is small, the gurgle always being above the umbilicus. This very exact meaning the sign does not possess, for it may be produced also in myasthenia, in the prolapsed as well as in the vertical stomach, when the border of the stomach is below the umbilicus, and may even be produced to the right of the median line, when the lesser cul-de-sac is flabby and displaced downward to the right—it being only necessary so to locate and shape the line of compression as to shut off a pouch containing gas, or gas and fluid. By the gliding method, the lower border of the stomach may sometimes be felt to slip from beneath the hand. It is unnecessary to state that this gastric palpation sign should not be confused with the movable gurgling ribbon. The gliding gurgle and the gliding palpation may be used to locate the lower limit of the stomach, when the very particular conditions for the realization of the signs are present.

Gastric clapping is produced by bringing the walls of the stomach separated by gas into contact by a simple, quick depression of the epigastrium in the median line. A somewhat similar sound may be elicited by clapping the anterior wall of the stomach against the surface of the liquid from which it is separated by a layer of gas: but in this condition gastric splashing may also be heard. As the word implies, it differs in its qualities from gastric splashing which, unlike clapping, can not be generated when the patient is standing. It is unnecessary to state that succussion can only yield splashing sounds. Gastric clapping, engendered during the period of physiological repose, is a sign which suggests, but does not establish, myasthenia. When it can be produced below the umbilicus, the clapping denotes that the stomach is either enlarged and myasthenic or is displaced.

Gastric splashing occurs in a stomach containing gas and fluid—it matters not whether the wall be flaccid or rigid. The sound may be generated in many ways—by motion of the trunk, by the rapid movements of the diaphragm, by the contraction and relaxation of the abdominal muscles, by alternating depression at two points, by compression at one point and tapping at another, or by three or four rapid depressions

over the stomach (particularly at the end of expiration) without raising the fingers from the abdominal wall. One method may succeed when the others fail to elicit the sound. The patient should lie flat on the back, with the muscles relaxed, and the stomach should not be distended. Gastric splashing may occur during the period of digestion. The stomach may then be normal. But constant splashing during the digestion of a meal should excite suspicion. The sign may be produced when the stomach should be empty, and is then always pathological, and reveals, according to the moment when the examination is made, either excessive secretion or stagnation or retention. Splashing may be absent during the period of physiological rest, and be elicited by none of the methods employed, even at the end of expiration. If half a glass of water be now given, and the sign be present, suspicion should be excited. After an interval of half an hour if the examination is again positive, there is myasthenia. If, after the use of the half-glass of water, none of the gastric noises can be elicited, the motor power of the stomach is normal. The area over which the splashing can be produced is also useful in locating the stomach and in detecting its displacement. But we must first be sure that the sound is intragastric.

Either a splashing or a clapping sound may be produced in the colon, containing a mixture of gas and fluid, or only gas. Constipation, so frequent in myasthenia, would make it probable that the sound is intragastric. Palpation of the colon may at once clear up the difficulty, for it is narrow and ribbon-like or cord-like, and rarely its contents may be solid. The area of distribution might correspond in location and somewhat in form with either the colon or the stomach. The sound first produced, after the drinking of the half-glass of water, is intragastric. In doubtful cases, the emptying of the stomach by the tube might locate the noise, but it is better to repeat the examination on the following day if the source of the sounds is not made clear. But the possibility of the colon and the stomach at the same time yielding clapping or splashing sounds should not be forgotten.

The area of the splashing, which should be marked out by using gently one or two fingers in producing the rapid, successive, vertical, and slight depressions in order to localize the effect, may reveal the size and the location of the stomach, and may thus enable us to discover or to exclude a displacement, a large myasthenic, or an obstructed organ. We may proceed in either of two ways. The splashing is most easily

excited and most constant over the triangle of contact of the stomach with the abdominal wall, bounded by the median line, the left costal border, and a line uniting the cartilages of the ninth rib. The cartilage of the ninth rib, which is one of the landmarks of the stomach, may be readily found by passing the finger, from below, along the costal border. The first notch is between the very movable tenth cartilage below and the less movable ninth cartilage above. Over this area the gastric splashing will be found. We proceed from this area downward and to the right, and unite points where the splashing disappears; or proceed from below, along parallel vertical lines, and from the right, along parallel horizontal lines, toward the region of the stomach, and mark and unite the points where the splashing begins. The lower limits may be more easily marked, when the patient's shoulders are slightly raised, and the physician steadies the stomach by gentle pressure over the pyloric region. To find the right limit, the patient may be turned very slightly on the right side. In the attempt to locate the stomach by direct percussion splashing it often happens that the sound is not produced immediately beneath the fingers, and the lower boundary of the stomach may be placed too high or too low. The sound is produced where the gas and fluid mix, and the lower part of the stomach may contain only fluid, or the fluid may all gravitate backward into the greater cul-de-sac, and leave only gas in the part of the stomach beneath the epigastrium; or, again, the finger agitation may be transmitted to the stomach through the medium of an adjacent organ. The tapping should be light, and the result controlled, where possible, by the gliding method of Glénard. To avoid the error due to the distribution of the contents of the stomach, the depressions with the fingers should be made near the line of the greater curvature, at the end of a deep inspiration, and, also, while the other hand of the physician, which is laid flat on the epigastrium, gently compresses the upper part of the stomach. It is a useful precaution to ascertain whether the boundary of splashing is changed after the administration of half a glass of water.

The constant absence of gastric splashing during the period when the stomach would be normally empty excludes myasthenia with certainty. The strong, healthy stomach does not splash during the period of digestion, or only splashes intermittently as the stomach momentarily relaxes. The complete absence of splashing occurs only when the stomach is very strong.

CHAPTER III.

THE FUNCTIONAL SIGNS.

THE clinical history and the physical examination may enable us to form a correct diagnosis without further exploration. The clinical history may be typical and the physical signs may be characteristic. But disease does not develop in grooves and yield always clear-cut types. We may even go further and truthfully say that in the large majority of the cases these methods leave us in doubt. How often, after a most painstaking history and a most exhaustive physical examination, we must be content with a probable diagnosis, or a mere guess at the truth.

This additional knowledge, so sorely needed, may be furnished in part by the functional signs. These signs possess both positive and negative value, inasmuch as certain persistent pathological variations may reveal a particular disease; and normal functions, or a particular functional anomaly, may exclude a disease suggested by the previous questioning and the physical examination.

But the functional signs are even more valuable in treatment, and have the great advantage of being direct and precise. They form the only scientific basis of the dietetic treatment, which has for its object the nourishment of the body and the favoring of the diseased organ. The stomach must work, and, this being the case, we must know what it is capable of doing before we ever can favor it in its duties or avoid overtaxing it. But this is not all. The functional signs display the dynamic variations, be they hypersthenic or asthenic, and consequently suggest and control the physiological treatment. Whether our remedies be foods or drugs, or of other nature, we must select them in accordance with the commanding physiological indications—excitation or sedation. But still more; a diagnosis made without a knowledge of the functional signs is always incomplete. The clinical history and the physical signs, in a small number of the cases of stomach troubles, may reveal the particular disease, such as ulcer, carcinoma, gastroptosis, gastritis, or one of the dynamic affections. The anatomical diagnosis, however, is insufficient. To treat the stomach well, its functional power must be known. Without such knowledge there can be no intelligent and organized effort to preserve, restore, and com-

pensate its functions. And we hold it a mistake in practice to substitute an inference, a mere guess, for the definite knowledge obtained by the functional exploration.

The functional signs are also helpful guides in prognosis, revealing how serious the trouble is when the first examination is made, and making clear, when the exploration is repeated, the gains and losses and the general tendency of the evolution of the disease of the stomach. It is correct practice to make a functional examination whenever this can be done without danger, and the functional signs are likely to prove valuable in diagnosis, prognosis, or treatment.

The stomach has three functions—general and special secretion, the churning and evacuating movements, and absorption. It also serves as a reservoir, the filling and emptying of which is roughly self-regulating.

The contents of the stomach, obtained during the period of functional activity and during the period of functional repose, yield functional, bacteriological, and anatomical signs. The functional signs reveal the functional power and activity of the stomach. They may be conveniently treated under the following divisions: (1) Secretion; (2) the motor function; (3) absorption; (4) digestive work.

1. SECRETION.

The specific secretions of the stomach are three in number —the acid, and two ferments. The other ferments claimed to exist by some physiologists possess at present no clinical importance. The general secretion of the stomach is the mucus. The secretions of the stomach are formed by its glandular lining membrane, which displays numerous longitudinal folds. Slightly magnified, the lining membrane appears reticulated, the little pits (several millions in number) being the mouths of the glands.

The surface is paved with a single layer of cylindrical epithelial cells, which also extend to a variable distance into the necks of the special secretion glands. Into the pyloric glands they extend four or five times deeper than into the glands of the fundus. The mucous glands are lined throughout with the cylindrical epithelium. These cells are long, thickly packed, and sharply limited, except at their concealed, deep extremities. The basal end is filled with a finely granular protoplasm up to near the central part where the nucleus lies. From the nucleus outward to the free or surface ex-

tremity, the cells contain clear mucus. This differentiation of the intracellular contents is made very clear by staining. During functional activity more and more protoplasm is transformed into mucin, the free extremity swells and bursts and discharges the mucus into the stomach. The cell retains

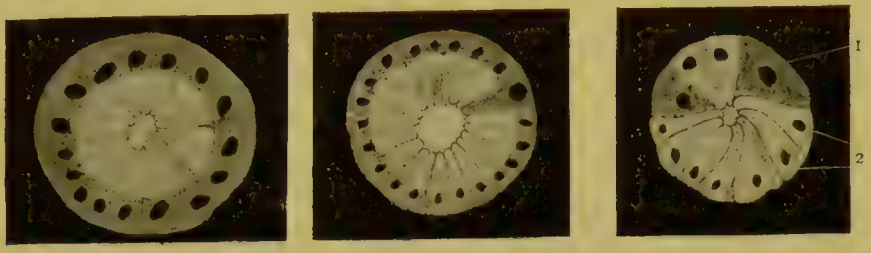


Fig. 4.—A, Cross-section of a mucous gland, × 325. B, Cross-section at the mouth of a peptic gland, × 325. C, Cross-section at the neck of a peptic gland, × 325: 1, border cell; 2, chief cells.



Fig. 5.—A, Surface epithelium, × 530. B, Border cell, × 530. C. Chief cells in repose (1) and in activity (2), × 530.

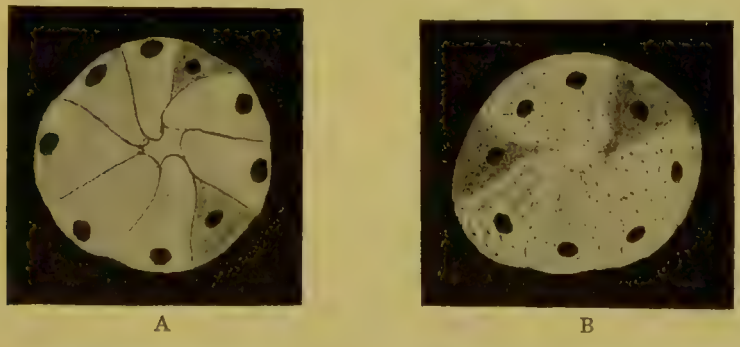


Fig. 6.—Cross-sections of the body of a peptic gland in repose (A) and in activity (B), × 325.

the unconverted protoplasm and the nucleus, and, presenting a goblet shape, begins its work anew. Mucus secretion is greatest in catarrhal gastritis, when the goblet cells are also found in large numbers. The mucus, which is the general secretion of the stomach, is formed by the cylindrical cells.

The special secretion glands contain two kinds of cells—the chief (adelomorphous) and the border (delomorphous) cells. The chief cells are small, cuboidal, mononuclear, badly defined, and filled with granular protoplasm, which has little affinity for the anilin dyes. The chief cells predominate in the base of the gland, are less numerous in the fundus, and are very few in number near the neck of the gland. They lie next to the lumen of the gland, are most numerous during the period of functional repose, and almost disappear at the height of secretory activity. The border cells are large, roundish, well defined, and stain deeply with the anilin dyes. The border-cells predominate near the neck of the gland, are found in

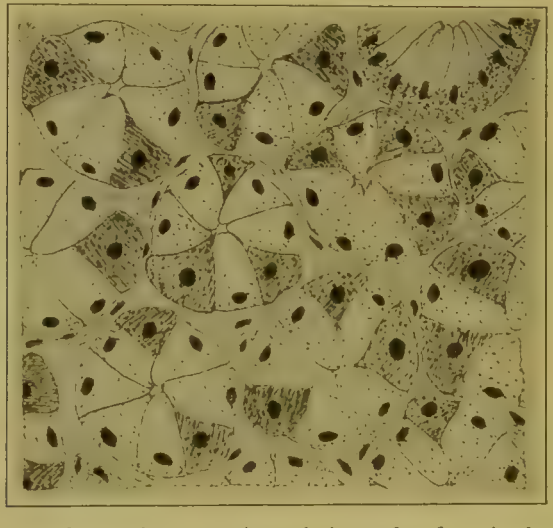


Fig. 7.—Cross-section of normal mucosa through the necks of peptic glands one hour after the test-breakfast, × 240 (authors' specimen).

about equal number as the chief cells in the fundus, and only here and there along the base. They lie near the membrana propria, and form only a small part of the wall of the lumen. The border cells are most numerous during secretory activity, and are very few in number after long fasting.

In the glands of the pyloric region, the chief cells are numerous, but only here and there is a border cell found. The border cells in the area about the junction of the pylorus and the body of the stomach differ slightly in their staining affinities from the border cells found in other parts of the stomach. The glands also vary in their size and form in the different parts of the stomach, some being tubular and some branching, some long and others short. The secretion of

the pyloric region is less acid, but contains more pepsin than that of the fundus of the stomach. But it is more than probable that the chief cells do not form the ferments exclusively, nor do the border cells form acid alone. The border cells undoubtedly form both acid and ferments, and it seems very likely that the border cells are developed from the chief cells. A study of development and of cell life, and the data of physiology and of pathology, make this theory plausible.

To test the secretory functions of the stomach we require: (1) A method of obtaining the contents of the stomach; (2) a test-meal to excite secretion in a normal manner, and (3) reagents and apparatus necessary for the analysis of the removed contents.

1. Methods of Obtaining the Stomach-contents.—Many efforts have been made to save the patient the disagreeable sensations associated with the use of the stomach-tube. Nature sometimes imperfectly does the work for us, when the patient vomits at the right moment during the digestive period, or in the early morning before any food or drink has been taken into the stomach. But the vomit may consist largely of the secretions accompanying or preceding the act, and manifestly can not be taken as an index of the specific secretion or the digestive activity conditioned by the disease alone. The vomit usually consists of the contents of the stomach mixed with mucus, with bile, with pancreatic juice, with duodenal secretion, and with saliva. Its analysis may give some valuable information, particularly if it occurs when the stomach should be empty, or at a certain interval after a meal. Continuous secretion, excessive secretion, the power and the degree of secretion, and the motor activity, may be suggested by the properties of the vomit when the vomiting occurs under certain conditions. But the information thus obtained must be complemented and confirmed by the usual functional tests. In no case can the examination of the vomit render unnecessary the test-breakfast, the test-dinner, the test-supper, or the morning expression and lavage. The examination of the vomit is valuable, but the act can not be agreeably produced at the will of the patient or of the physician, and it occurs under exceptional circumstances.

Some of the very laudable endeavors to avoid the employment of the tube show a good deal of ingenuity. Einhorn in 1890 proposed as an easy method the use of a little silver, olive-shaped stomach-bucket attached to a strong thread. The thread is attached to an internal valve-like lid, which closes the mouth when the thread is tight, and leaves it open when

the thread is relaxed by the bucket reaching the gastric contents. The inventor, however, now attaches the thread to a ring on the inside of the bucket, and claims that on account of the bucket being full when withdrawn, the contents are not likely to become mixed with other secretions. The bucket is carried into the stomach by a big swallow, remains there a while, and is then drawn out by the thread and pulled over the larynx while the patient makes an effort to swallow. The small amount of contents is tested for free HCl and for ferments.

Edinger had already (1880–81) proposed a similar method, the principle of which is as old as Spallanzani. Small pieces of sponge are freed from alkaline carbonates by treatment with hydrochloric acid, and by washing with distilled water until the reaction is neutral. The dried compressed sponge, to which a silk thread is attached, is inclosed in a gelatin capsule, the thread being drawn through a perforation in the cover. The capsule is next smeared with a little butter, swallowed, and washed into the stomach with a bite of bread and some water. The capsule is dissolved in the stomach, the sponge is uncovered, and after fifteen minutes it is quite rapidly pulled out, saturated with the gastric contents. The four or five drops thus obtained were tested for free HCl. The capsule may be swallowed, according to our object, while the stomach is empty, or at a certain time after a meal.

Späth (1887) recommended a little bulb of elder pith, saturated with a solution of Congo-red, to be swallowed, and thus dipped into the contents, and withdrawn, the blue coloration revealing the presence of free acid.

The methods of Späth, of Edinger, and of Einhorn are hardly more pleasant than the employment of the stomach-tube. Einhorn's bucket furnishes enough contents for a limited qualitative analysis.

The Sahli-Günzburg method (1889–91) was extensively used. About three grains of iodid of potash are tied watertight in a very small, thin piece of rubber tissue by means of a thread of fibrin. Günzburg made the knot with the fibrin thread, while Sahli brought the two ends of the fibrin filament together and tied them close up with a fine thread. Günzburg administered the capsule one hour after the test-breakfast; Sahli gave it with the test-breakfast. The trimmed packet is inclosed in a gelatin capsule, swallowed at the desired moment, and the saliva is frequently tested for iodin with strongly acidulated starch paper, which is absorbed

after the digestion of the fibrin string, and is eliminated by the salivary glands. The packet may be opened by the intestine instead of by the stomach, or in the stomach by organic acids, or even by chlorids or salines.

Compared with the use of the tube, none of these ingenious but very limited methods can be seriously deemed more than an elegant amusement. The functional tests should be thorough, sufficient, and as free as possible from error, or



Fig. 8.—The extremities and openings of several stomach-tubes now in usé: *a*, Ewald; *b*, Riegel; *c*, Hayem; *d*, *e*, Van Valzah and Nisbet.

they should be omitted altogether. For the proper exploration of the functions of the stomach, the stomach-tube is absolutely necessary.

The stomach-tube has long been employed in therapeutics to wash out the stomach, or to introduce food or remedies. As a diagnostic instrument, it is used to detect obstruction of the cardia, to locate the lower border of the stomach by palpation of its extremity when it is introduced, to inflate

the stomach with air, to determine the duration of gastric digestion, and to remove the contents of the stomach at any desired moment. The employment of the stomach-tube as an instrument of diagnosis and research has made the once obscure subject of gastric pathology one of the longest and the clearest of internal medicine.

Many varieties of tubes are now in use. A good one should be soft, perfectly smooth, with a closed, well-rounded, somewhat conical lower end, and stiff enough to allow the slight resistance to its introduction to be overcome without its curling up. It should possess one velvet-eye opening of the same size as the caliber, and another very small one with similarly depressed or rounded edges. The large and lower opening should be as near the extremity as possible, and the caliber of the tube should end with it. The small upper opening, on a level with and opposite the upper border of the large opening, diminishes the chances of the tube becoming obstructed or tearing away a piece of the mucous membrane by aspiration. With two large holes the risk of obstruction is twice as great, and the closing of the upper hole cuts off the outflow. The two large openings weaken the tube, and the stomach can be emptied no lower than the higher one; but the mucous membrane is prevented from being caught by suction during aspiration or siphonage and is thus protected against injury.

The caliber of the tube should be as large as it is possible to make it, and yet retain the essential elasticity and stiffness. The length should be 75 to 90 cm., the longer ones being useful in cases where the greater curvature of the stomach is low. It should be kept as scrupulously clean as a catheter, and each patient with syphilis, tuberculosis, or cancer, should have one for his exclusive use.

The Indications for the Use of the Tube.—Experience alone can point out those cases in which an exploration of the functions of the stomach is likely to prove of most value. The tube should be used in every case where the clinical diagnosis can not be made without it, and where no contra-indication to its employment exists. Without the use of the tube the functional power of the stomach can only be inferred, and the inference is usually far from the truth. An exploration of the functions of the stomach is never useless and is rarely unnecessary, but we are frequently able to treat cases successfully, although not in the best manner, without it.

1. The tube may be used to make the diagnosis.
2. The tube may be used to confirm the diagnosis.

3. The tube may aid in reaching a diagnosis by exclusion.
4. The tube may be used to obtain valuable information in prognosis and treatment, and to determine what progress has been made.

Contraindications to the Use of the Tube.—The introduction of the tube is a little operation which may need to be abandoned on account of the general condition of the patient, or on account of the presence of some particular disease rendering it dangerous or injurious. The contraindications may be multiplied by the timidity or the prejudices of the physician. The contraindication is absolute when the operation is a menace to life; it may be advisable or best to avoid it under other exceptional conditions. The common sense and the experience of each physician should form rules for his own guidance.

In the severe acute diseases of the throat and the stomach, in general peritonitis and in perigastritis, its employment is contraindicated. Advanced soft carcinoma and ulcer, on account of the danger of hemorrhage or perforation, prohibit absolutely the introduction of the tube, as do also a similar condition of the esophagus and the existence of aneurysm of the aorta. It is best to defer the operation in all cases of recent hemorrhage. Old age, adynamic diseases, uncompensated valvular disease of the heart, degeneration of the heart muscle, arteriosclerosis with a past hemorrhage, advanced renal disease, cyanosis, and all troubles in which injury is likely to result from a slight shock, from increase of blood pressure, or from obstructed respiration, certainly make it advisable to avoid the use of the tube, except in very unusual circumstances. When in doubt, it is best to give the patient the benefit of it. The tube should rarely be introduced during pregnancy, and never if there is a history of previous abortion. These general rules may sometimes be violated without inflicting injury, and the refusal of the patient to permit the use of the tube may render a search for contraindications unnecessary.

Introduction of the Tube.—(*a*) *The Difficulties of Introduction.*—The first difficulty most likely to be met with is an irritable throat, a very frequent accompaniment of chronic gastritis. If the patient by intelligent co-operative effort and regular breathing can not overcome the choking, the pharynx should be anesthetized with cocain.

An obstruction may be encountered in the esophagus, due to stricture, pocketing, compression, tumor, swelling, spasm, or to a foreign body.

The most common difficulties, however, are faulty manipulation on the part of the physician, and the resistance of the patient, who should be told what to do. A little knowledge may dissipate all fear. It is useless to try to force a soft tube through a contracted pharynx, and an effort to do so is very likely to result in the curling up of the tube in the throat, or in its passage into the larynx.

(*b*) *The Methods of Introduction.*—The finger method is the older one, but it is better suited to the introduction of bougies or of stiff esophageal sounds. The index- and the middle fingers of the left hand are introduced into the right side of the mouth, and the base of the tongue is pressed downward and forward. The tube, held as a pen, and grasped by the right hand about six inches from its extremity, is pushed along the left index-finger and directed slightly to the right side of the throat, so as to avoid irritating the epiglottis. The tube is next pushed on, as the right hand is elevated by the side of the posterior pillar into the esophagus. The operation is done rapidly and gently. As soon as the tube is well inserted in the esophagus, the fingers are taken out of the mouth and the instrument is pushed rapidly into the stomach, the patient being told to breathe deeply. It is far better to let the patient direct the tube into the esophagus by swallowing. The fingers commonly excite choking, resistance, and fear.

The technic of the swallowing method is very simple: The patient sits erect, and bends the head slightly forward, with the chin a little elevated; all the clothing is loose, and both hands are left free or engaged in holding the receptacle for the contents. The tube, wet simply in warm water, is held in the right hand, as a pen, while the index-finger and the thumb of the left hand protect the lips and steady the tube in the median line. The tube is now placed on the tongue as far back as the base, the patient is told to swallow, and as the pomum adami rises, the progress of the tube is aided by a gentle push. The procedure introduces the tube into the esophagus, and as the patient breathes quietly the instrument is pushed rapidly on into the stomach. The sudden yielding of the slight resistance offered by the cardia marks the entrance of the tube into the stomach.

But this resistance may not be felt, and it may be wise to locate the cardia before beginning the introduction of the tube. This can be done by marking on the tube the average distance of the cardia from the incisor teeth. The esophagus in the adult is about 25 cm. long, and extends from the

lower border of the inferior constrictor muscle of the pharynx behind the cricoid cartilage and on a level with the fifth cervical vertebra to the cardiac orifice of the stomach. From its beginning it turns to the left of the trachea, and passes behind the left bronchus on its way to the abdominal cavity through the esophageal foramen, ending about three cm. below the diaphragm in the cardiac orifice of the stomach. The distance of the cardia from the incisor teeth is about 40 cm. —15 cm. to the beginning of the esophagus, 5 cm. cervical, 17 cm. thoracic, and 3 cm. abdominal. About 5 cm. more should be added so as to bring the tube well within the cavity of the stomach; or the distance from the incisor teeth to the cardia may be measured by placing the lower end of the tube over the tip of the process of the ninth dorsal vertebra, and running the tube along the spine and the side of the neck to the front teeth. To express the contents, the openings of the tube should be placed just within the cardia, or below the level of the contents. To remove the contents by siphonage, the internal end of the tube should extend to the greater curvature.

After the tube is introduced, it should always be held in position, in order to prevent its accidental complete entrance into the stomach. To remove the tube, first draw it through the cardia, compress it tightly between the thumb and index-finger of the right hand, and complete the removal as the thumb and index-finger of the left hand catch the internal extremity.

The Removal of the Stomach-contents.—The contents of the stomach may be obtained in three ways: (1) by suction; (2) by self-expression; (3) by position and gravity.

1. *Suction* is the oldest method (Jukes). For this purpose the pump is antiquated and the modified Politzer bag of Ewald or the aspirator of Boas (Fig. 9) should be used.

The aspirator of Boas consists of a compressible rubber bulb, terminating at each end in a short rubber tube, which may be compressed by the fingers or closed by clamps. One end is attached, by means of a short piece of glass tubing, to the stomach-tube. The end communicating with the stomach-tube is clamped and the air driven out through the other end by compressing the bulb; the outer end is next clamped and the communication with the stomach-tube re-opened, or the end may be fitted with a valve. The contents of the stomach are drawn into the bulb, and expelled by compression, after clamping the stomach-tube end, into the receiving-glass. Strauss recommends a very excellent apparatus. It

consists of three pieces of rubber tubing fitted with clamps, attached to the extremities of a T-shaped piece of glass tubing. One of the tubes is attached to the stomach-tube, another to a funnel, and the third to two bulbs like those of a double bulb atomizer. By proper manipulation the stomach can be washed out, inflated with air, or emptied. Gross inserts a glass bulb between a rubber bulb and the stomach-tube; the glass bulb receives the contents, and to it is attached a manometer.

An attempt to completely empty the stomach by suction should not be made, but only enough of the contents should



Fig. 9.—Boas' aspirator.

be removed to serve for an examination. The suction should be let off, and some water allowed to flow in, before the removal of the tube. This method should be used whenever the contents of a degenerated or ulcerated stomach are removed, or when the patient is weak or has advanced disease of the lungs. Expression would here be more dangerous. In cases, also, where there is so much mucus in the stomach as to prevent expression, aspiration may be employed.

2. *Expression* is quick, and requires no additional instrument except a glass to receive the contents. The patient has only to take a deep inspiration, hold it, and contract the abdominal muscles, or make efforts to cough, or to strain as at

stool. If the patient can not or will not co-operate, the tube may be moved back and forth through the cardia, so as to excite an effort to vomit.

3. In many cases the contents of the stomach can be more easily and completely removed by placing the patient in the horizontal or knee-elbow position. There is then no uphill curve to the tube, and the fluid contents are easily voided with a little increase of abdominal tension. The tube should be just through the cardia, and held in the mouth so as to prevent its dragging forward on the larynx. The method is very valuable in completely emptying the stomach, and is very easy and efficient when the patient is accustomed to the tube.

Sometimes no contents can be obtained. The failure may be due to several causes: The tube may be obstructed: in this case it can sometimes be opened by forcing a little air through it. But the obstruction may not be removable by this procedure, and the tube must be withdrawn, and again introduced after its canal is opened. The tube may be introduced too far, and its extremity may be curled up above the level of the stomach-contents: This fault may be remedied by withdrawing the tube a few inches. The stomach-contents may be too thick or too coarse for removal, or the stomach may be empty: The situation may be cleared up by introducing a little water and then withdrawing and analyzing the fluid obtained.

2. The Test-meals.—The exploration of the functions of the stomach should be made under conditions as nearly alike as possible to those under which the viscus ordinarily does its work. Food is the physiological excitant of these functions, and a test-meal furnishes a natural, simple, practical, and agreeable excitant.

Gastric secretion may be excited by electricity, but it is not practicable to use this agent at the bedside to test even secretion. Thermal and chemical excitants once met with some favor, when it was an object to obtain the secretion of the stomach unmixed with food. Leube, after washing out the stomach, introduced 50 c.c. of a three per cent. solution of soda, and allowed it to remain in the stomach twelve minutes. At the expiration of this period 50 c.c. of lukewarm water were introduced, and the mixed contents were withdrawn. The reaction of the mixture should, normally, be neutral. If the reaction is alkaline, the degree of alkalinity represents the degree of insufficiency of secretion. Leube also introduced the ice-water method. One hundred c.c. of ice-water were

introduced through the tube, and at the end of ten minutes 300 c.c. of water were introduced, and the whole contents immediately withdrawn. The liquid was tested with litmus and tropæolin for acids, and for ferments by artificial digestion. Jaworski improved the method by introducing 200 c.c. of ice-cold distilled water and removing it without dilution. The method is no longer employed by the great clinician who first used it.

The test-meals proposed are very numerous. The white-of-egg test-meal was recommended by Jaworski. The patient is put on an albuminous diet for a few days before the testing is begun. In the meantime, the state of the stomach in the morning before breakfast is learned by aspiration with the tube. If the exploration is negative and the stomach is found empty, 100 to 300 c.c. of distilled water are introduced and withdrawn. The fluid withdrawn is saved for analysis.

The evolution and phenomena of digestion are next studied, after the patient has taken into a clean and empty stomach the white of one or two hard-boiled eggs, with 100 c.c. of distilled water at the temperature of the room. After the patient has remained quiet for forty-five minutes, he is allowed to drink 100 c.c. of distilled water, and five minutes later the contents are aspirated through the tube. This is saved for analysis. The stomach is further washed until all the white of egg is removed and the undissolved part is recovered by filtration and compared with the quantity eaten. On a second morning the test is repeated, but the aspiration is made after ninety minutes. The normal stomach contains no white of egg after the expiration of seventy-five minutes, all having been digested or evacuated undigested into the duodenum, except, possibly, a few pieces caught in the folds of the mucous membrane. The maximum of digestive activity is attained in thirty to forty minutes. The first aspirations from the fasting and digesting stomach are tested for reaction, free HCl, digestive power, mucus, syntonin, and for peptones, both qualitatively and quantitatively. Morphological elements are looked for with the microscope.

The method of Jaworski is an excellent one, but it also has its disadvantages. The white of egg appeals directly to the unique secretory work of the stomach, but it constitutes a very restricted meal. Klemperer recommended a pint of milk and two small rolls. Bourget recommends 20 gm. of well-browned dry toast, 150 c.c. of weak tea without sugar, and a teaspoonful of essence of peppermint. But the test-breakfast of Ewald and Boas, the test-meal of Germain Sée,

and the test-dinner of Riegel have best stood the test of time. These three test-meals meet all requirements.

The Test-breakfast of Ewald and Boas.—The patient is given in the morning on an empty stomach, one roll (about 70 gm.) and 350 c.c. of water or weak tea (about 1½ glasses). It is recommended that the bread be taken into the mouth dry, be thoroughly masticated, and, after insalivation, washed down with the water or tea. Incomplete mastication delays the digestion of the crust, and the particles are very liable to obstruct the tube. One hour after the beginning of the breakfast the contents are removed.

This breakfast contains albumin, starch, sugar, fat, extractive matter, and inorganic salts, is rarely repulsive, easily expressed, and suitable for chemical analysis. The roll weighing 70 gm. contains about 5 gm. of proteids, 39 gm. of carbohydrates, ⅓ of a gm. of fat, and ¾ of a gm. of ash. Normally, at the end of an hour we obtain from 30 to 50 c.c. of a yellowish-tinged, homogeneous mixture, filtering with ease. The total acidity is about 55, the acid albumin acidity about 45, and the free HCl acidity about 10. These figures represent the number of cubic centimeters of a decinormal solution of potash or soda required exactly to neutralize 100 c.c. of the stomach-contents. The digestive power of the filtrate (Hammerschlag's test) is about 90 per cent. The filtrate, in a dilution of 1 : 3000 with $\frac{1}{20}$ normal HCl solution, digests albumin after remaining in the thermostat at 37° C. for twenty-four hours. Labferment coagulates milk in dilution of 1 : 40, and labzymogen in dilution of 1 : 160. Free HCl appears in thirty minutes, reaches its height in about one hour, and, diminishing, continues to the end of gastric digestion. Acetic acid and ferrocyanid of potassium give a little cloudiness after the first half-hour, and the reaction is demonstrable to the end of digestion. The biuret reaction (rose) runs the same course. Fehling's solution is reduced during the first hour, and then the reaction is less pronounced, and disappears entirely in one-half to three-quarters of an hour before the end of digestion. Lugol's solution gives a brownish-purple color during the first one and one-half hours. The contents contain no blood, a small quantity of mucus, possibly a little bile forced through the pylorus during the expression, and a small number of micro-organisms. No organic acids are present, unless they have been swallowed or set free from their salts by the HCl of the gastric juice. The total acidity is equal to the HCl-albumin-acidity, plus the free HCl, plus the small quantity of acid phosphates

which are usually present. The stomach is empty in from two to two and one-half hours after the beginning of the breakfast. The following table displays the evolution of the hydrochloric acidity during the normal digestion of the test-breakfast:

	Thirty Minutes.	Sixty Minutes.	Ninety Minutes.
Total acidity,	20 to 30	50 to 60	30 to 40
HCl-albumin,	20 to 30	40 to 50	25 to 35
Free HCl,	0	10 to 15	5 to 10

The Test-meal of Germain Sée.—The test-meal of Germain Sée consists of 60 to 80 gm. of chopped beef, free from fat and fibrous tissue, 100 to 150 gm. of white bread, and a glass of water. It is best to give definite quantities, and we use the smaller quantities in order to get a meal which demands more work of the stomach than does the test-breakfast and much less than does the test-dinner. This meal contains about 20 gm. of proteids, 1½ gm. of fat, 56 gm. of carbohydrates, and one gm. of ash. The contents are removed two hours after the beginning of the meal, which should not be eaten rapidly.

After the test-meal of Germain Sée, about 40 to 60 c.c. of grayish-yellow contents are obtained at the end of two hours. The total acidity ranges between 50 and 70, the free HCl between 10 and 20, and the HCl-albumin between 40 and 50. With Hammerschlag's test the digestive power of the filtrate is about 90 per cent. In dilution of the filtrate (1 : 3000) with $\frac{1}{20}$ normal HCl solution, albumin is digested after standing twenty-four hours in the thermostat at 37° C.; labferment is active in dilution of 1 : 40, and labzymogen in dilution of 1 : 160. Lugol's solution gives a brownish-violet coloration. Acetic acid and ferrocyanid of potash produce a light cloud. Fehling's test reveals a moderate quantity of sugar. Very few muscular fibers and starch granules are discoverable. Free HCl first appears in one and one-fourth to one and one-half hours, and is present in a mere trace near the termination of gastric digestion. The biuret reaction is positive (rose-colored) between the first and third hours, and then disappears. During the same period acetic acid and ferrocyanid of potash produce cloudiness. Both these tests are negative during the last fourth of digestion. Fehling's solution is negative during the last half-hour of digestion. The stomach is empty in three and one-half hours after the beginning of the meal. The following table displays the acidity at the end of each hour:

	One Hour.	Two Hours.	Three Hours.
Total acidity,	30 to 40	50 to 70	30 to 40
HCl-albumin,	30 to 40	40 to 50	25 to 35
Free HCl,	0	10 to 20	0 to 10

The Test-dinner of Riegel.—The test-dinner of Riegel consists of a plate of beef soup, 150 to 200 gm. of beefsteak, 50 gm. of purée of potatoes, and a small roll. For uniformity and accuracy, we prescribe 300 c.c. of clear beef broth, 150 gm. of beefsteak (fillet), 50 gm. of mashed potatoes, and 35 gm. of white bread. This meal contains about 36 gm. of proteids, 3.5 gm. of fat, 30 gm. of carbohydrates, and 5 gm. of ash. The food should be thoroughly masticated, and the tough, fibrous pieces of the steak should be removed. The contents are obtained either three or four hours after the beginning of the meal.

The quantity of contents obtainable ranges from 40 to 70 or 80 c.c. The total acidity varies between 60 and 80, the HCl-albumin between 50 and 60, and the free HCl between 10 and 20. Free HCl first appears in about two and one-half hours, continues about two hours, and disappears about twenty minutes before the stomach becomes empty. The biuret reaction (rose) and the cloudiness with acetic acid and ferrocyanid of potassium begin near the end of the first hour, and disappear during the last fourth of the period of gastric digestion. Very few striated muscular fibers can be found. The stomach should be empty at the expiration of five hours from the beginning of the meal. The ferments and the starch products are present in the same strength as in the test-meal of Germain Sée. The following table makes clear the evolution of hydrochloric acidity :

	Two Hours.	Three Hours.	Four Hours.
Total acidity,	40 to 50	45 to 70	60 to 80
HCl-albumin,	40 to 50	45 to 60	50 to 60
Free HCl,	0	0 to 5	10 to 20

The test-breakfast, the test-meal, and the test-dinner reveal, accurately and fully, the secretory activity and the digestive work of the stomach. They also afford a rough estimate of its motor activity. But each one of the tests has its advantages and disadvantages. The test-breakfast requires only one hour's delay, and, furnishing the stomach a light task, displays the secretory activity of the stomach in an ideal manner. The chemical analysis of the contents after the test-breakfast is easy. But it does not fully test the digestive power of the stomach, and the demands made on the stomach

are much less than those of the usual daily meals. To test the functional sufficiency of the stomach, the test-meal of Sée or the test-dinner of Riegel is necessary. Indeed, it is wise to use two or three of these tests in order to avoid errors, and we employ them, in the study of our cases, in the order of the increasing demands which are made by them on the functions of the stomach. The information obtained in this manner suggests to the experienced clinician what further tests are advisable, and whether it is necessary to search for a disorder in the normal evolution of digestion.

1. The Hydrochloric Acid.

Before proceeding to study the gastric contents, a very important physiological and practical question must be answered—viz., Is hydrochloric acid formed by the cell and given out in a free state?

All authors do not give the same answer to this question. Some maintain that it is secreted in organic combination with leucin or pepsin. Others believe that the hydrochloric acid is formed by lactic acid out of the chlorids. These theories may be dismissed without further consideration.

Recently another theory has been advocated, which maintains that the free hydrochloric acid found in the gastric contents is a by-product of digestion. The chlorids, according to this theory, are the physiologically active constituents of the secretion, these being formed in the cells and given out by them chiefly as the chlorid of sodium. Working in unison with the pepsin they combine with the proteids, and in so doing may form free hydrochloric acid. According to this theory of Hayem, the free hydrochloric acid in the contents is not a cellular but a chemical and digestive by-product. This view we can not accept.

The almost universally accepted theory—and in our opinion the true one—claims that the hydrochloric acid is formed and given out in a free state by the border cells. This secreted hydrochloric acid unites with the mucus, the exfoliated cells, the saliva found in the stomach, and the inorganic carbonates, etc., of the food and saliva; and this part, for the digestion of the food, is lost; another part unites with the proteids of the food and converts them into acid compounds, which, with the aid of pepsin, are built up into albumoses and peptones. After the affinities of the proteids are satisfied, the secreted hydrochloric acid remains free. The hydrochloric acid, then, is secreted free, and remains free only after

it is in excess. This excess is limited by the normal stomach, and when this limit is exceeded or not reached within the proper time for the particular test-meal, the acid secretion is abnormal.

Is it possible to estimate the quantity of HCl secreted? If it be remembered that this secreted acid combines almost immediately with inorganic bases, with organic bases, and loosely with proteids; that the stomach discharges its contents into the duodenum intermittently throughout the period of digestion, forms a variable quantity of mucus, exfoliates a variable number of cells, has received a variable amount of bases capable of being drawn away from their union by this strong nascent mineral acid; that the stomach also probably absorbs sufficiently to vitiate the results—it will be seen that the estimation of the total amount of HCl secreted is surrounded by many difficulties. Moreover, the quantity of saliva secreted in twenty-four hours varies from 200 to 2000 gm.; this is all swallowed and enters the stomach. The saliva contains inorganic salts in the proportion of 2.24 to 1000, nearly all of which are composed of the chlorids and carbonates in the proportion of about four parts of the former to one of the latter. The gastric secretion also contains 0.2 per cent. of chlorids, and a variable quantity is introduced with the test-meals. Of the inorganic chlorids found in the gastric contents after a test-meal, a variable part is formed of combinations with the secreted HCl and a variable part comes from other sources. It is not practicable to estimate the total quantity of HCl secreted during the digestion of a meal, but it is possible to estimate the total quantity found at a particular moment in the stomach which is physiologically active, and, knowing the total acid-combining power of the proteids in the particular test-meal, to estimate roughly the total quantity of the secreted HCl which has been used in the digestive transformation of the food.

The secreted HCl in inorganic combination is lost to gastric digestion, but it protects and influences intestinal digestion. The union of the acid with these substances is strong, and all these affinities are satisfied before the HCl combines loosely with the proteids, before its digestive work begins, before it remains free. Consequently, the HCl which combines with the proteids and that which remains free together roughly represent the activity of acid secretion. The albumin-combined HCl represents the actual digestive work. We know that the combined and unused HCl should be found in

certain quantities in the normal stomach in the very particular conditions in which the test is made. We therefore conclude that the quantity of HCl loosely combined with albumin, together with the quantity remaining free in the contents withdrawn at the end of a particular time after the eating of a particular meal, is a practical and clinical measure of the secretory activity of the peptic glands, and of the digestive work of the acid of the stomach.

The variations from this clinical standard can not be attributed exclusively to disease of the border or chief cells, which may or may not be normal. The healthy stomach possesses what may be designated as its acid sense, by which the hydrochloric activity is held fast to a line marking the normal evolution of acid secretion in keeping with the physiological action of the contents. This special sense may be disordered; the nerve-centers concerned with the secretion may generate, and receive or send out morbid impressions; the circulation may not be good; the blood may be impure or poor; the border cells themselves may be diseased—and one or all of these conditions may be expressed by the hydrochloric acidity. The acid signs are of great value in detecting an anomaly; but only with the greatest circumspection should they be made the revealing signs of a particular disease.

To estimate the hydrochloric acidity of the stomach-contents, removed either during fasting or during the digestion of a test-meal, is not very difficult. The details of the methods can be mastered by study and practice. No very great skill is required; neither is it necessary nor expected that the clinician should be an expert chemist. But if it be necessary to appeal to chemistry, the analysis should be exact and possess a precise meaning.

Some of the many methods proposed for the chemical examination of the gastric contents are fit only for the laboratory. Their finer distinctions and greater exactness make them preferable in scientific research. But a clinical method must be simple and short; otherwise the demand upon the skill and the time of the practitioner will prevent its general use, and will deprive it of practical value. Any long and complicated method will fall into disuse, or will, at best, be generally deemed a mere whim, peculiar to a few doctors. A description of the more complete and exact methods should find a place in a special work of this kind. The useless or erroneous methods require no mention. The simple clinical methods which require little time or skill will usually be found sufficiently exact and complete.

We have seen that the hydrochloric acid secreted by the cells of the glandular lining membrane of the stomach may be found in four states in the contents of the digesting stomach:

1. Free, when secreted in excess of the affinities of the organic and inorganic matter found in the stomach.
2. Combined with the proteids—a loose, acid-reacting, digestive combination.
3. Combined with organic bases.
4. Combined with inorganic bases, forming chlorids. These inorganic chlorids subtract from the acidity due to secretion just so much as the chlorin contained in them represents. The inorganic chlorin, drawn from the hydrochloric acid secreted, escapes estimation, and is lost to digestion.

The general acidity of the contents of the digesting stomach is not fully represented by the acidity due to the hydrochloric acid, which is free and combined with albumin. Other factors enter and make it more complex. The general acidity may be due to—

1. Free and albumin-combined hydrochloric acid.
2. Free organic acids, which may also be in acid-reacting combination with proteids, provided no free and stronger acid is present.
3. Acid inorganic salts, chiefly the acid phosphates.

The last two factors of general acidity are introduced into the stomach or are formed there without the aid of the specific secretion. The organic acids are chiefly the products of micro-organisms in the stomach, and will be carefully studied with the bacteriological signs. The functional diagnosis is concerned only with the first of these factors—the hydrochloric acidity. We will now describe how the free and albumin-combined hydrochloric acid in the mixture represented by the gastric contents can be detected and estimated. The analysis is qualitative and quantitative.

Qualitative Tests.—The qualitative analysis, which often suffices for practical purposes, is made with color-reagents.

The gastric contents may be alkaline, neutral, or acid. If they are neutral or alkaline, further testing for hydrochloric acid would naturally be useless. Consequently, as a first qualitative test we use a reagent sensitive to all free acids, organic acid combinations, and acid salts. The best reagent of this kind is a good quality of litmus. The red litmus paper is rarely required in this analysis, as an alkaline reaction of the contents of the digesting stomach is exceedingly rare. The blue litmus paper is so sensitive that a neg-

ative result is at once final. A good quality gives a plain reaction with 0.006 per cent. hydrochloric acid, 0.01 per cent. lactic acid, and 0.02 per cent. butyric acid. This blue dye, united with an alkali, is soluble in water, and is sensitive to the hydrochloric acid combined with albumin, this mineral acid having a stronger affinity for the alkaline base of the dye than for albumin. The blue litmus is consequently reddened by all the factors of general acidity.

The second acid qualitative test is made with Congo-red. This is a red dye, soluble in water, and is colored blue by all free acids. It is three or four times more sensitive to free hydrochloric acid than to the free organic acids of the gastric contents. The intensity of the blue coloration increases with the percentage of free acid, and organic acids produce a muddy grayish-blue or purple, in contradistinction to the pure blue of the mineral acid. But even a very extended experience should not permit these slight distinctions to be valued as more than suggestions. The hydrochloric acid and other acids combined with albumin or organic bases, do not alter the color of Congo-red. Acid phosphates, in very concentrated solution, produce a brownish coloration; but in such quantities as are found in the stomach after a test-meal, produce no change of color. Consequently, the Congo-red is turned blue by the free hydrochloric and the free organic acids. The acid salts and the acid organic combinations are without influence. A positive reaction means the presence of a free acid. Congo-red may be used in aqueous solution or in the more convenient form of paper. The latter reacts plainly with 0.01 per cent. free hydrochloric acid and with three times the quantity of lactic acid. The solution is about ten times more sensitive than the paper, which is made by saturating fine filter paper with an aqueous solution of Congo-red. After it is dry, the paper is cut into narrow strips of convenient length. The test may be made with the solution by spreading a few drops over a white ground,—as in a watch-glass, on a piece of white paper, or in a porcelain crucible,—and allowing a drop of the gastric contents to flow over it from the edge; the area of contact becomes blue. Or the paper may be dipped into the mixed contents, after the ordinary manner of using test-papers.

The third qualitative test, in case the Congo-red gives a positive result, is for free hydrochloric acid. The surest and most sensitive reagents for this purpose are those of Günzburg and Boas.

The reagent of Günzburg is an alcoholic solution of phloroglucin and vanillin:

Phloroglucin,	2 gm.
Vanillin,	1 gm.
Alcohol (absolute),	30 gm.

This reagent is uninfluenced by all acid salts and combinations and by free organic acids. As hydrochloric is the only free mineral acid present in the gastric contents, unless other mineral acids have been swallowed, a positive reaction with phloroglucin-vanillin is proof of the presence of free hydrochloric acid. This regeant is also very sensitive. With a solution of 1 : 10,000 the very fine crystals appear, while a solution of 1 : 20,000 gives a red coloration. If there be much organic matter in the contents, the red is mixed with the pasty, dry, yellowish residue. The test is best made by placing three or four drops of the reagent in a porcelain crucible, and spreading it by causing it to flow in different directions over the surface. A like quantity of the filtered gastric contents to be tested is now added, and spread over the same area. With a small flame the crucible is slowly warmed, never allowing it to become too hot to be comfortably borne on the back of the hand. After several seconds a clear red coloration appears, or the fine, bright-red crystals may be seen if free hydrochloric acid be present. The phloroglucin-vanillin paper, recommended by some writers, we do not employ. A few drops of the gastric contents are placed on the paper, which may be more rapidly heated in the crucible than with the solution test.

The following solution of Boas' is used in the same manner as the Günzburg reagent:

Resorcin (resublimed),	5 gm.
White sugar,	3 gm.
Alcohol (95 per cent.),	100 gm.

The coloration is a bright rose-red. This reaction of the gastric contents is characteristic of free hydrochloric acid. It is very sensitive, and may be even clearer than the Günzburg reaction when the contents contain much soluble albumin.

Dimethylamidoazobenzol, methyl-violet, and tropæolin 00 are also employed as free hydrochloric acid reagents.

A very sensitive reagent for HCl is a ½ per cent. alcoholic solution of dimethylamidoazobenzol. This yellowish solution changes to a reddish color on the addition of a mere trace of free HCl. The reaction is positive with an HCl

solution of 1 : 20,000. The reaction is also produced by concentrated solutions of acid phosphates and lactic acid, but such concentrations occur so rarely and in such particular conditions as to allow little chance for an error to be made by any one who is familiar with the analysis of the gastric contents.

Methyl-violet is changed to a sky-blue color by free HCl. The intensity of the blue color produced varies with the strength of the hydrochloric acid solution—1 : 10,000 gives a bluish tinge, and 1 : 5000 a clear reaction. But the reagent is not so sensitive when the gastric contents are employed instead of an aqueous solution of HCl. Lactic acid, in a solution of 1 : 300, produces also a bluish tinge. Chlorids also vitiate the results, and the methyl-violet, consequently, is not altogether satisfactory as a free HCl test. The test, which is a very pretty one, is performed in the following manner: A test-tube full of a very dilute solution (1 : 500) in distilled water is prepared (a clear violet color), and two small test-tubes are about half filled with it. To one is added one to three c.c. of the filtered gastric contents, and to the other the same quantity of distilled water. By comparing the two tubes, the change of color is beautifully displayed.

Tropæolin 00, in concentrated aqueous solution, is an excellent reagent for detecting free acids, but it is not quite so sensitive as Congo-red. The yellow solution is changed to a deep red by free acids. Used in the following manner, it is a sure reagent for free HCl (Boas): Three or four drops of a saturated alcoholic solution are spread thinly in a porcelain crucible, and over the same area an equal quantity of the filtered gastric contents is allowed to flow. Next, heat slowly over a small flame. If free HCl is present, lilac streaks appear near the border, which, on further heating, become blue.

Whenever free HCl is present in the gastric contents, it is useless to make a test for HCl in combination with proteids, since the HCl remains free only when the acid affinities of the proteids have already been satisfied. The presence of HCl free proves that HCl in combination with proteids is also present.

But it often happens in the diseases of the stomach that no free HCl can be detected. Under such circumstances, if the reaction of the contents is acid, HCl in proteid combination may be present. To detect it, qualitatively, two tests may be made, one of which is a chemical and the other a color test. A small quantity of the filtered contents is ex-

actly neutralized, boiled, treated with acetic acid and sodium chlorid, again boiled, and filtered after cooling. Any albumin left in the filtrate is digested albumin combined with HCl. A positive biuret reaction (rose) on treating the filtered contents with liquor potassa and cupric sulphate shows the presence of propeptones. These chemical tests also give roughly the degree of peptonization. The color test is made by employing a one per cent. aqueous solution of alizarin. Three or four drops of the solution of the dye are added to a small quantity of the filtered contents, and decinormal alkaline solution is added until a pure violet color appears. To a second portion of the filtered contents the same quantity of the alkaline solution is added. If the mixture still reacts acid to litmus, HCl in proteid combination is present. For practical qualitative purposes the biuret test is sufficient.

The Quantitative Analysis.—On account of its bearing on the diagnosis and treatment of the diseases of the stomach, an easy and an accurate method of differentiating and estimating the different factors of the acidity of the gastric contents is very desirable. For a long period the chemical analysis was confined to the detection of free hydrochloric acid. Hayem and Winter (1888) proved the insufficiency of this method, and gave their very valuable, but long, chlorometric analysis. A new light was turned on the chemical pathology of the stomach. Many other quantitative methods have since been given, but none of these seem completely to satisfy the requirements of practice. The busy physician in his daily work demands a quantitative method at once easy, accurate, and rapid.

It would seem that the practical value of very slight quantitative variations in the factors of general acidity has been overestimated, and that the simpler color methods reveal with sufficient exactness all the deviations from the normal chemism which possess a distinctive practical meaning. The inaccurate laboratory methods need not be mentioned. The more complete and chemically accurate methods—which may sometimes be used with advantage in practice, and which should always be employed in original research—will be given. But for daily needs coloration-titration procedures usually suffice.

(*a*) **The Color Methods.**—*The Method of Mintz.*—This method estimates the quantity of free HCl, or H, by means of Günzburg's reagent. To ten c.c. of the filtered contents, the decinormal alkaline solution is added from the buret until the reaction of a droplet (platinum loop) of the fluid with the

reagent of Günzburg becomes negative. If the reaction is still positive with 0.9 c.c., but is negative with 1.0 c.c. of the titration alkali, the free HCl acidity in 100 c.c. of the gastric contents is represented by 10 c.c. of the decinormal solution of caustic soda or potash. One c.c. of a decinormal alkaline solution represents 0.00365 HCl; consequently, the 100 c.c. of the gastric contents contain ten times that amount. The strong free HCl in the contents is first completely neutralized by the alkali before any of the other factors of the total acidity are affected. The method consumes less time if the platinum loop, wet in the solution, is brought in contact with Congo-paper. When the Congo-red is no longer made markedly blue, the use of the more trustworthy reagent of Günzburg may be begun.

The Method of Boas.—The object of this method is the quantitative estimation of the free hydrochloric acid, or H. Five c.c. of a watery solution of Congo-red are added to an equal quantity of the filtered contents. The mixture becomes blue. The titration is made with the decinormal solution of caustic potash or soda, and contin ueduntil the original Congo-red color is restored. As a control color, five c.c. of the Congo-red solution may be added to an equal quantity of distilled water. The titration should be slow near the end, as the restoration of the red color does not take place rapidly.

The value thus found represents free HCl; or, more accurately, free HCl and the quantity of free organic acids present, or H + O. The author of the method claims that the quantity of organic acids in the contents after the test-breakfast is practically seldom worth considering when free HCl is present, but that if the organic acids are present in notable quanity, they should be removed by repeated shaking with ether before the titration.

Method of Töpfer.—The method of Töpfer requires the following color reagents: (1) One-half per cent. alcoholic solution of dimethylamidoazobenzol; (2) one per cent. aqueous solution of alizarin; (3) one per cent. alcoholic solution of phenolphthalein.

1. To ten c.c. of the filtered contents are added a few drops of the alcoholic solution of phenolphthalein, and the total acidity is titrated with a decinormal solution of caustic potash or caustic soda. The alkali is to be added until the rose-red coloration is permanent, and drop by drop so long thereafter as the rose color does not become deeper. The end reaction of the color indicator is used. This gives the total acidity, or A.

2. To ten c.c. of the filtered contents are added three or four drops of a ½ per cent. alcoholic solution of dimethylamidoazobenzol. This yellowish solution is changed to red by a trace of free HCl, being as sensitive as the reagent of Günzburg. Organic acids (free) produce the red color when in a concentration of 0.5 per cent. and in the presence of mucin and albumoses require even a greater concentration. Such a quantity of organic acids is probably never present in the free HCl-containing contents (qualitative tests) of the test-breakfast, given on a clean and an empty stomach. The titration is made with the decinormal alkaline solution until the original light orange (not lemon yellow) replaces the red color produced on the addition of the dimethylamidoazobenzol to the contents. The value thus obtained represents the free HCl, or H.

3. To ten c.c. of the filtered contents are added three or four drops of the one per cent. aqueous solution of alizarin. This reagent is sensitive to all the factors of gastric acidity except the organic combined HCl, or C; or, in other words, the alizarin solution becomes pure violet on the addition of the solution of caustic alkali after all the factors of gastric acidity have been neutralized except C, which does not prevent the transformation of color. This solution, which is yellowish, becomes brownish, and then pure violet, as the titration proceeds. As soon as the *pure violet color*—which is the same as that produced by a one per cent. solution of sodium carbonate—appears, the titration is complete. This value gives the free HCl and the acidity due to the acid salts and the organic acids, or H + P + O, A — (H + P + O) = C. A — (H + C) = O + P. (A, total acidity; H, free HCl; P, acid salts; O, free organic acids; C, HCl combined with proteids.) The quantity of the decinormal alkaline solution used in each titration is multiplied by 10 to bring all to the standard, which is 100 c.c. of the gastric contents; or it is better to use only five c.c. of the filtered contents, and to multiply the results by 20. The method, after practice, gives good results, but the proper use of the alizarin as an indicator requires care and experience.

Combination Color Method.—The color methods may be combined in such a manner as to make the quantitative estimate of the factors of acidity of the gastric contents sufficiently complete and accurate for the requirements of practice.

1. If the qualitative tests have revealed the presence of free

hydrochloric acid in the gastric contents, we may proceed as follows:

A qualitative test for free volatile organic acids should be made. An acetic or a rancid odor would create suspicion. A small quantity of the contents is put into a test-tube, and a strip of moistened blue litmus paper is held in the end of the tube while its contents are gently warmed. The litmus paper will be reddened if a volatile acid is present. Lactic acid in the contents of the stomach containing free hydrochloric acid has no distinctive pathological significance, and if found was probably introduced as such, or was set free by decomposition of lactates in the test-breakfast or in the saliva. On the other hand, acetic and butyric fermentations are frequent in the presence of free hydrochloric acid.

Ten c.c. of the filtered or well-mixed unfiltered contents are placed in a beaker, and the titration, with a decinormal solution of caustic potash or of caustic soda, is begun. The level of the solution in the Mohr buret is noted on a piece of paper after reading the mark of the lowest part of the meniscus, the finger being placed just below the point so as to make the demarcation clearer. The decinormal alkaline solution is allowed to flow until, after shaking, a drop taken out (conveniently with a platinum loop) gives a negative reaction with a few drops of a ½ per cent. alcoholic solution of dimethylamidoazobenzol or with Günzburg's reagent. The point where the reaction with these color reagents disappears gives, when read on the buret, the quantity of decinormal alkaline solution required to neutralize the free HCl contained in the ten c.c. of the contents. A memorandum of this reading is made on a slip of paper. This gives H, or the quantity of free HCl.

A droplet of the contents (platinum loop) is now brought into contact with a few drops of an aqueous solution of Congo-red. If the reaction is positive (grayish-blue), free organic acids are present. The titration is continued with the solution of Congo-red as indicator. As soon as the reaction fails, the reading of the buret is again taken, and the additional quantity of decinormal solution used represents the quantity of free organic acids. Thus O is obtained.

A droplet of the contents (platinum loop) is next brought into contact with a few drops of a one per cent. aqueous solution of alizarin. If the reaction is negative, the titration is continued with the alizarin as indicator. The point where the pure violet reaction begins marks the disappearance of the acid salts. The reading on the buret is again taken, and a note is made of it. This gives P.

The remaining acidity is titrated after the addition of a few drops of a one per cent. alcoholic solution of phenolphthalein. The decinormal alkaline solution is added until there is no longer an increase of the rose-red color; not the beginning but the end reaction is taken to indicate the completion of titration. The level of the solution is again read on the buret. The additional quantity of decinormal solution used represents the acidity due to HCl in organic combination. This is C.

The total quantity of the decinormal solution used represents the total acidity of the ten c.c. of the analyzed contents.

It only remains to calculate the acidity represented by each factor in 100 c.c. of the gastric contents—the quantity taken as the convenient standard of comparison. Ten c.c. being used in the analysis, the results are all multiplied by ten. This gives the total acidity and the different factors of it, expressed in so many cubic centimeters of decinormal solution.

After experience with the color reagents, the analysis can be done rapidly without using, in making the repeated tests, a sufficient quantity of the contents materially to falsify the results. The procedure may be easily controlled by repeating the analysis, or part of it, on a fresh specimen.

2. If there be no free HCl but free organic acids in the contents, as indicated by the Congo-red, the test is made as before for volatile acids. If the volatile acids are absent, the free acidity is due to lactic acid. If volatile acids are present and Uffelmann's reaction is negative, the free acidity is due to acetic or butyric acid, or to both. The reactions for lactic and volatile acids may both be positive, as in obstructive retention in carcinoma, with absence of free HCl. The titration is begun with Congo-red as the indicator, and completed with alizarin and phenolphthalein as before. This gives O, P, and A. C may be incorrect, on account of the possible union of organic acids with the proteids.

3. If no free acid is present, as indicated by Congo-red, the titration begins with alizarin and is completed with phenolphthalein. This gives P, C, and A; C being again possibly incorrect.

If the contents contain no free HCl, the deficiency of HCl secretion may be roughly determined by the method of von Noorden and Honigmann. Decinormal solution of HCl is added to ten c.c. of the filtered contents until the reactions for free HCl appear. The quantity of the decinormal solution of HCl represents the deficiency in the secretion of hydrochloric acid. This test is of more value when made on the

unfiltered contents. Congo-red and Günzburg's reagent should be used as the indicators. If free organic acids are present, these should be estimated first and neutralized. The test is then made by using ten c.c. of the unfiltered contents whose free acidity has been neutralized. The decinormal solution of HCl is added until dimethylamidoazobenzol (droplet method) or Günzburg's reagent reveal the appearance of free HCl. The quantity of the decinormal HCl added has been used in the formation of acid proteid compounds and in the displacement of organic acids. Consequently, after the free HCl reaction becomes positive, the quantity of organic acids set free must be estimated by further titration with a decinormal alkaline solution until Congo-red no longer gives a reaction. The quantity of alkali used should be subtracted from the quantity of decinormal HCl obtained in the first part of the titration. If no HCl has been secreted and combined with proteids, the two titrations will give the same quantities, all the decinormal HCl being utilized in the displacement of organic acids.

(*b*) **The Chemical Methods.**—*The Method of Braun.*—The method of Braun is one of the simplest of the chemical methods. The general acidity (A) of ten c.c. of the filtered contents is estimated by titration with a decinormal potash or soda solution, using phenolphthalein as the indicator.

To a second ten c.c. of the filtered contents, in a platinum crucible, is added a decinormal solution of potash or soda in excess of the general acidity as given by the first analysis, and this quantity is noted. The alkaline fluid is next evaporated carefully on an asbestos plate, and the residue is incinerated, care being taken not to heat the crucible beyond a dull red glow, and to stop as soon as no more points are in ignition. The ashes are next dissolved with a quantity of decinormal H_2SO_4 (or HCl) solution equal to that of the decinormal potash or soda solution added before evaporation. The solution is next warmed to drive off the CO_2, and titrated with phenolphthalein and decinormal KOH, or NaOH. This gives the total acidity, with the exception of that due to the burnt organic acids, or $H + C + P$. $A - (H + C + P) = O$. For example, ten c.c. of the filtered contents require six c.c. of the decinormal K(OH) to neutralize it, or 60 per 100 c.c. This gives $A = 60$ decinormal K(OH). To a second ten c.c. of the contents are added seven c.c. decinormal K(OH); this is then evaporated, incinerated, and seven c.c. decinormal H_2SO_4 (or HCl) added, and the dissolved ash, after heating, requires five c.c. decinormal K(OH)

for neutralization. C + H + P = 50 decinormal K(OH); 60 — 50 = 10 = O, or the organic acids converted into alkaline carbonates by incineration and combined, with the equivalent acid added. This method gives O (C + H + P) and A.

The Method of Hayem and Winter.—The method of Hayem and Winter is very long, but when well carried out is also very accurate for H + C.

In three porcelain capsules, a, b, and c, are placed five c.c. of filtered gastric contents.

To the capsule a is added an excess of pure carbonate of soda, and then the contents of the three capsules are slowly evaporated to complete dryness on the water-bath. The capsule a, to which the carbonate of soda was added, contains all the chlorin of the gastric juice in the form of inorganic chlorids. The capsule a gives the total chlorin, or T. The capsule is next brought to a low red heat, slowly and frequently stirring with a glass rod, so as to avoid loss by little explosions. As soon as no more points are in ignition and the carbonate of soda begins to fuse, the incineration is completed. After cooling, the residue is taken up with distilled water, to which a little pure nitric acid has been added. The solution, which should be clear, is next boiled to drive off the CO_2. It is then completely neutralized, or rendered slightly alkaline with pure carbonate of soda. Heat until an abundant precipitate falls, taking down the carbon with it. Then filter, wash the precipitate with boiling water, unite all the washings, and estimate the quantity of chlorin with decinormal $AgNO_3$ in the presence of neutral chromate of potash. One or two drops of a concentrated solution of neutral chromate of potash are added to the filtrate, and the decinormal solution of nitrate of silver is allowed to flow into it from the buret until the red coloration, after shaking, remains permanent. All the chlorin is now combined, and the silver begins to unite with the chromic acid. The titration is complete. The number of cubic centimeters of the decinormal silver solution multiplied by 0.073 (20 × 0.00365) expresses in terms of HCl the quantity of chlorin contained in 100 c.c. of the gastric contents.

The capsules b and c, by the prolonged and complete evaporation at 100° C., have been deprived of their HCl (free). To the capsule b we add an excess of carbonate of soda, and fix the remaining chlorin. We proceed as with the first capsule, and estimate the quantity of chlorin: a — b = H, or free HCl.

The contents of the capsule c are incinerated without the

addition of carbonate of soda. The process is rapidly done by heating on a wire gauze while breaking the coal with a glass rod. As soon as the coal is dry and friable, the process is complete. After cooling, treat as capsule a. This capsule contains only the fixed inorganic chlorids. The free HCl has been driven off and the combined HCl (C) destroyed by heat: b — c = C, or organic combined HCl; a = T; a — b = H; b — c = C; c = F, or inorganic combined chlorin.

This is a chlorometric method. The values found may be converted into their HCl equivalents, or multiplied by 20 to give the equivalent quantities of decinormal $AgNO_3$ required to combine each chlorin factor in 100 c.c of gastric contents.

In the analysis of Hayem and Winter, the quantity obtained for b (C + F) is incorrect. Not all the free HCl is driven off by evaporation, and the heating causes more of the free HCl which is present to combine with the proteids. But a (= T) and c (= F) are correct. T — F = H + C. In case no free HCl is present, T — F = C. If the contents contain free HCl, the quantity should be estimated by Günzburg's reagent, or by Töpfer's dimethylamidoazobenzol, which would give H.

The Method of Lüttke.—Like that of Hayem and Winter, the method of Lüttke is chlorometric. The total chlorin (T) and the chlorin combined with inorganic bases (F) are quantitatively estimated. The difference, or T — F, represents the physiologically active chlorin (H + C)—*i. e.*, the quantity of secreted HCl left free (H) and combined with proteids (C).

The chlorin is estimated by the method of Volhard. For this purpose are needed: (1) A decinormal acid solution of pure nitrate of silver and (2) a decinormal solution of ammonium sulphocyanid.

The decinormal acid silver nitrate solution is prepared by the following formula:

Argenti nitras (c. p.),	17.5 gm.
Acidum sulphuricum (25 per cent. solution),	900. c.c.
Liquor ferri sulphurici oxidati,	50. c.c.

Mix and dissolve in the above order, and add enough distilled water to make one liter. Correct by using a standard decinormal solution of HCl. Ten c.c. of the silver solution are measured and diluted to 100 or 150 c.c. with distilled water. This dilution is titrated with the decinormal solution of HCl. If, for example, 9.5 c.c. of the HCl solution are required to exactly combine the silver in the 10 c.c. taken, 950 c.c. of

the acid silver solution are diluted to 1000 c.c., and the correction is confirmed by a new titration.

The decinormal solution of the sulphocyanid of ammonium is prepared by adding eight gm. of NH_4CNS to a liter of distilled water, and the solution is corrected by means of the decinormal silver solution. If, for example, 9.8 c.c. of the decinormal silver solution are required to produce the first light rose color, persisting after shaking, 980 c.c. of the solution are diluted to 1000 c.c., and the correction confirmed by titration, until the quantity of CNS in one c.c. of the one is just sufficient exactly to combine the quantity of Ag in one c.c. of the other.

If the acid silver solution is added in excess to the gastric contents, only so much of the silver is precipitated as chlorid of silver as there is chlorin present to combine with it. The unchanged nitrate of silver is estimated by titration with the decinormal solution of ammonium cyanid, after removal of the precipitated chlorid of silver by filtration. When the cyanid is added to the acid solution containing the nitrate of silver and sulphate of iron, cyanid of silver and the cyanid of iron are formed.

$$AgNO_3 + NH_4CNS = AgCNS + NH_4NO_3.$$
$$Fe_2(SO_4)_3 + 6NH_4CNS = Fe_2(CNS)_6 + 3(NH_4)_2SO_4.$$

The cyanid of iron colors the solution blood-red, but so long as $AgNO_3$ is present, $Fe_2(CNS)_6$ is decomposed and AgCNS formed, and the red color disappears. The first persistence of a rose color indicates that all the nitrate has been converted into the cyanid of silver, and the quantity of the decinormal solution of ammonium sulphocyanid used represents the quantity of silver nitrate unconverted into the chlorid of silver.

1. *The Analysis.*—Ten c.c. of the *well-mixed unfiltered* gastric contents are placed in a 100 c.c. graduate, a, and the measure graduate is washed out a number of times with distilled water, the washings being poured into the large graduate. Twenty c.c. of the decinormal acid solution of nitrate of silver are added, the mixture is well shaken, and left standing for ten minutes. All the chlorin contained in the specimen combines with the silver and forms the insoluble chlorid. Other combinations of silver are prevented by the presence of the H_2SO_4. Next, add enough distilled water to make the mixture measure exactly 100 c.c., and filter, using dry paper, a dry funnel, and a dry beaker. Fifty c.c. of the filtrate are titrated with the decinormal solution of ammonium sulpho-

cyanid, the titration being complete as soon as the reddish color persists after shaking. The reading on the buret is taken, and gives one-half of the silver nitrate added *in excess*. The reading, multiplied by two and subtracted from 20, gives the total quantity of chlorin present in the ten c.c. of the gastric contents. If, for example, six c.c. of the titration fluid have been used, six multiplied by two and the product subtracted from twenty, gives eight c.c. to represent the total chlorin, or T; 0.00355 multiplied by eight, or 0.0284 gm. of chlorin, in the ten c.c. of gastric contents, or $0.00365 \times 8 = 0.0292$ gm. of HCl. Thus T is estimated.

2. A second ten c.c. of the mixed unfiltered contents are placed in a platinum crucible, b, and evaporated (best on an asbestos plate). The residue of evaporation is incinerated by holding the crucible directly in the flame until the organic matter no longer burns and there is a dull red glow. The incinerating should be done rapidly and without overheating, as high, prolonged heat would also decompose the inorganic chlorids. The free and combined HCl (H + C) has been driven off by the evaporation and incineration, and the ash contains only the inorganic chlorids.

Pathologically, large quantities of ammonium chlorid (as in uremia, putrefaction, etc.) and, normally, traces of this salt, are found in the contents of the stomach. The ammonium chlorid is decomposed and driven off by incineration, and thus escapes estimation. The quantity of inorganic chlorids will be just as much too small, and the remainder, T — F or H + C, too great, the ammonium chlorid being included in the estimate of T. This is a possible source of error, which does not exist with T and F in the method of Hayem and Winter.

The ash is next dissolved by rubbing repeatedly with hot water, the extraction fluid being emptied upon a filter. About 200 c.c. of hot water are required, and a small quantity of the last washing should give no precipitate with the silver solution. The whole of the filtered washing, after the addition of ten c.c. of the decinormal silver solution and filtration, are titrated with the decinormal solution of ammonium cyanid, and the value found is subtracted from ten. The remainder is the value for the inorganic chlorids (except chlorid of ammonium, if it should have been present). Thus F is estimated. If, for example, seven c.c. of the titration fluid were used, seven subtracted from ten gives three: $0.00355 \times 3 = 0.01065$ gm. chlorin, or $0.00365 \times 3 = 0.01095$ gm. free HCl in the ten c.c. of gastric contents.

The total acidity, A, is estimated by phenolphthalein. The

free acidity, B, is estimated by using a ten per cent. solution of tropæolin in dilute alcohol as the indicator, in the same manner as phenolphthalein, the yellow mixture changing to red as soon as the titration is completed. $T - F = H + C$. $A - (H + C) = O + P$. If $A = T - F$, then $B = H$. If $A > T - F$, organic acids are present. If $B = A - (T - F)$, all the HCl is combined, and all the organic acids are free. If $B < A - (T - F)$, all the HCl and a part of the organic acids are combined; the free acidity, B, consists wholly of free organic acids. If $B > A - (T - F)$, free organic acid and free HCl are present, all the organic acids being uncombined.

Many other chemical methods have been employed. These either have only a historical interest, or possess no advantages over the three methods already given. We recommend the method of Braun for the estimation of the organic acids, or O. The method of Hayem and Winter gives the most accurate results for T and F, and consequently for $T - F$, or $H + C$. The free HCl, or H, should be directly estimated by Günzburg's reagent. The total acidity, or A, is estimated by using phenolphthalein as the indicator.

The knowledge of the percentages of HCl in the contents is usually sufficient. But the total quantity of physiological HCl, or $H + C$, in the stomach at a given moment gives some further information concerning the activity of secretion and of the motor and absorptive functions. The absolute quantity of HCl in the stomach at the time of the removal of the contents may be easily calculated from the analysis of a part, provided the whole quantity of the contents is known. The total quantity of the contents can be determined by the acidity method of Mathieu and Rémond or by the specific gravity method of Strauss.

The Diagnostic Value of the Variations of HCl.—By some students the chemical types revealed by the analysis of the gastric contents after a test-meal have been considered distinct diseases. These diseases have been carefully described, and each of them has been supplied with a characteristic etiology, symptomatology, evolution, and treatment. Others give the chemical types a place among the complications, and speak of the various anatomical diseases complicated by this or that chemical variation. Neither of these opinions can be successfully defended, as the chemical types are only revealing signs or symptoms.

Furthermore, these chemical signs have no pathognomonic meaning. Neither a dynamic affection, nor cancer, nor ulcer, nor gastritis, are constantly associated with a particular varia-

tion. Hayem contends that in all gastric troubles with persistent chemical types there are anatomical changes, and often serious lesions of the mucous membrane; that the work of the stomach is compromised, like that of other organs, only when there is an anatomical disease. That the persistent chemical types have often a physical basis in histological pathology is, without question, true. That such a basis always exists is more than doubtful, for normal secretion depends not on the integrity of the gastric glands only, but also on a normal nerve- and blood-supply, on a normal motor function, and on normal absorption. The mucous membrane may be perfectly normal, and the secretion of HCl be abnormal and this abnormality may be the symptomatic expression of a disease which is not located in the stomach. But Hayem has rendered an inestimable service in searching for the physical basis of the chemical types, and by controlling the functional chemical signs by the revelations of autopsies.

That none of the chemical signs are pathognomonic, is no evidence against their diagnostic value, which may be both positive and negative.

1. The analysis of the gastric contents removed after a test-meal may give a normal quantity of free and combined HCl (H + C), or the quantity may be variable, notably increased or diminished, or the physiological HCl may be entirely absent.

If the quantity is normal, and if this has been proven by two, or, better, by three tests, made with two or three days' intervals, there is no anatomical disease of the glands of the stomach. The condition of the blood, of the nerve-centers controlling secretion, and of the secreting glands, is such as to allow the performance of the normal work. There may be myasthenia or neurasthenia gastrica, but it is more probable that the trouble of which the patient complains is located in the intestines or some other organ. Normal digestive chemistry excludes with certainty an extensive disease of the mucous membrane of the stomach, and may be an important sign in the differential diagnosis of the diseases of other organs from one another and from the diseases of the stomach. These may be autotoxic, reflex, cerebral, spinal, or may be due to disorders of the circulation or to the quality of the blood. Under the circumstances, the acid secretion will at times be normal, and at other times abnormal. Variable abnormal types are common in chronic gastritis, with acute exacerbations. Any form of variable chemism may be found in complicated myasthenia.

Excessive secretion of HCl is a sign of adenohypersthenia

gastrica, and indicates the employment of sedative medication and the protection of the mucous membrane against the irritating contents. This is a frequent symptom of chronic glandular gastritis, or it may be associated with a complicated myasthenia. In myasthenia it may exist as an expression of the irritation produced by the prolonged sojourn of the contents in the stomach; in a further stage of the same trouble there may be continuous secretion, and the stomach may be unable to obtain physiological rest. In the irritative stage of acute mycotic gastritis, and during the acute exacerbations of chronic gastritis, excessive acidity may manifest the glandular irritability. It is also the most common chemical sign of ulcer, or of its associated gastritis. Excessive secretion may occur as a dynamic affection, as in the crises of cerebrasthenia, tabes, myelitis, intestinal auto-intoxication, and uricemia, and may rarely be a symptom of carcinoma engrafted on an old ulcer.

Hydrochloric subacidity is a common sign of stages or forms of acute and chronic asthenic gastritis, of carcinoma, sometimes of ulcer, and of a large number of diseases of other organs, of the blood, and of disorders of the circulatory system. It is an asthenic sign, and may indicate the employment of excitant treatment.

Hydrochloric anacidity is exceedingly rare as a symptom of a dynamic affection of the stomach, but is somewhat common in forms of gastritis and in atrophy. Carcinoma rarely runs its course without the appearance of this sign.

The analysis of the acidity of the contents removed at the acme of digestion of the test-meals reveals one form of the quantitative variations of the hydrochloric acid secretion. But secretion may also be disordered in its evolution.

In order to detect the abnormal evolution of gastric digestion, it is necessary to extract the contents at various intervals during the digestion of the test-meal. For the test-breakfast, the intervals should not be longer than half an hour, while the extraction should take place from hour to hour when the test-meals of Sée and Riegel have been given, removing each time, preferably by aspiration, only enough of the contents for analysis; or the same test-meal may be given on successive days and the contents be removed, by expression, after increasing intervals, until the evolution of digestion is completely displayed. A test-meal should never be given when the stomach is not empty, and gastric retention renders it imperatively necessary to first employ thorough lavage, which may have to be repeated for several days in succession until

the secretory irritation due to retention subsides. This stringent rule must be observed with special care when investigating the evolution of digestion.

The evolution of secretion may be more rapid than normal. Expression after one hour may give only a small quantity of contents advanced in digestion and a somewhat less than normal acidity. The stomach is almost empty and digestion is in its decline. The evolution has been more rapid than normal, and the tube, introduced twenty minutes after the test-breakfast, or one hour after the test-meal of Sée, or two hours after the test-dinner of Riegel, will show that at this early period the secreted hydrochloric acid remains free. This rapid evolution of secretion is the expression of a morbid irritability or excitability of the glands, accompanied by the rapid evacuation of the contents of the stomach; or the rapid evolution of digestion may be due solely to the rapid emptying of the stomach, as may occur in incontinence of the pylorus, in forms of scirrhus, in some cases of chronic asthenic and chronic atrophic gastritis, and in hypermotility. Secretion ceases too soon because the stomach becomes empty too rapidly. Consequently, the too rapid evolution of digestion may be accompanied by hyperchylia, by hypochylia, or by achylia, but the abnormally rapid evolution of secretion is the expression of morbid activity of the glands and it may be manifested by hyperchlorhydria or by hyperchylia. Abnormally rapid secretion never occurs in adenasthenia, but it may occur when the duration of digestion is short, or normal, or long.

The evolution of secretion may be prolonged, and the prolongation may be due to excessive secretion, to continuous secretion, to myasthenia, or to obstruction of the pylorus or duodenum. All three stages of digestion may be prolonged,—the rise, the stationary period, and the decline,—being long, but regular, in their general characters. This disorder of evolution is due to excessive secretion; or the period of decline may be abnormally long on account of the failure of secretion to subside as the stomach evacuates its ingested contents, the lines representing the evolution of digestion being regular: the first two stages of digestion may be long or short, and accompanied by normal or excessive hydrochloric acidity. This disorder of evolution which is manifested by prolonged or continuous secretion is due to chronic proliferating glandular gastritis. Prolonged digestion may be due to excessive or to continuous secretion; it is never produced by adenasthenia; or prolonged digestion may be due to motor insuffi-

ciency, and the evolution of digestion will show sudden rises and falls, which display the irregularity of the evacuation of the contents of the stomach. (For the disordered evolution of secretion and digestion due to myasthenia and to obstruction, see the chapters on these diseases.) The lower the specific gravity of the contents, the greater is the proportion of gastric juice in the mixture. The specific gravity of the filtrate of the normal contents, one hour after the test-breakfast, varies from 1010 to 1015; and the specific gravity of the gastric juice is 1004 to 1006. In excessive secretion, the specific gravity of the contents is less than 1010; and when secretion is diminished, the specific gravity of the contents one hour after the test-breakfast, provided there be no myasthenia, is near 1020.

The evolution of secretion may be delayed. The contents removed after one hour show no free HCl, or a mere trace of it. After the expiration of two hours (test-breakfast), the quantity of the contents removed is larger than normal, digestive products are comparatively abundant, and the free HCl and combined HCl are both greater than given by the standard after one hour. There is myasthenia, or obstruction, with irritation from stagnation.

The functional signs furnished by the variations of the acid secretion should not in themselves be given too distinct a diagnostic meaning, but should be considered in connection with the other diagnostic signs. Used in this way, their diagnostic and therapeutic importance and value become at once apparent.

2. THE FERMENTS.

The gastric juice contains two ferments, the quantity and the quality of which vary in the diseases of the stomach. These variations can only be roughly detected and estimated, but when there is an extreme and constant deficiency or excess the examination gives most valuable information concerning the anatomical state of the glandular layer. Slight persistent quantitative variations may suggest the direction in which the disease is making its inroads.

The great diagnostic value of the quantitative estimation of the labferment and of labzymogen, and of pepsin and pepsinogen, is not admitted nor utilized by all clinicians. As a result of careful clinical study, we wish to emphasize the great practical utility of a knowledge of their quantitative variations.

The secretion of hydrochloric acid is, probably, the work of the border cells; but this biological work is conditioned

and influenced by so many circumstances as to make it impossible and erroneous to attribute the variations of this secretion to diseases of the cells themselves. The quantitative variations of the hydrochloric acid may be as great in the dynamic affection as in the anatomical diseases. This secretion is, besides, intermittent, and called forth by special excitation at recurring intervals.

The chief cells perform their biological work in a different manner. The secretion of the mother substances of the two active ferments displays the activity of the cell itself. The formative work, being continuous, is less directly the expression of digestive influences. Consequently, we do not find clinically, at least with the tests now employed, notable and persistent variations which are purely dynamic, or which are due to a disturbance of the circulation, like passive congestion. Clinically, a persistent deficiency and an excess, revealed by the tests now in use, are found only when the cells are diseased.

The form, however, in which the ferments are found is due to circumstances. The conversion of the mother substance into the active ferment is dependent on the presence, in the stomach, of the chemical reagents capable of producing the change. Consequently, it would be a mistake to attribute the variations of the quantity of pepsin and labferment to imperfect work of the chief cells. But we must consider the deficient formation of the mother substances, of which the ferments are the converted products, as evidence of disease of the cells, which are concerned in their production. The ferments may be absent when their prototypes are present in normal quantity. In such a case we must look for an explanation of the abnormality elsewhere than in the ferment-secreting cells.

(*a*) **The Labferment and Labzymogen.**—Labzymogen is probably a specific secretion of the chief cells, which by the action of weak free acids and of calcium chlorid is converted into the active labferment. The ferment is rapidly destroyed in alkaline fluids, but the mother substance remains intact. The presence of calcium salts promotes and seems essential to the milk-curdling action of the ferment.

The labferment coagulates milk by the disintegration of the casein. The coagulation differs widely from that of acids, and takes place in the presence—but independently—of any free acid in the stomach. The coagulation which is produced *en masse*, and without change of the reaction of the mixture, occurs when the medium is neutral or weakly acid. The

ferment coagulum contracts on standing, and separates the whey, which contains the ferments, and can produce coagulation in a fresh specimen. The most favorable temperature for the action of the ferment is between 33° C. and 44° C., but in the presence of chlorid of calcium it may take place at 20° C. A temperature of 70° C. destroys the ferment, but not the mother substance. Boiled milk is coagulated more slowly than uncooked milk.

The Qualitative Tests.—To five c.c. of sweet milk (Leo), three or four drops of the unfiltered and unneutralized gastric contents are added, and the covered glass is placed in the thermostat at blood-heat. If the labferment is present, the coagulation occurs in ten or fifteen minutes. A negative result with this method, on account of the very small quantity of the gastric contents used, should not be considered conclusive. In the short interval, coagulation by micro-organisms is hardly possible. But the following test is more conclusive:

Five c.c. of the filtered contents are exactly neutralized by the decinormal alkaline solution, always at hand, and added to an equal quantity of sweet neutral, or amphoteric milk. The glass is then placed in the thermostat at blood temperature. In from five to twenty minutes coagulation will demonstrate the presence of the labferment.

The qualitative test for labzymogen requires a special preparation of the gastric contents. Five c.c. of the filtered contents are made very slightly alkaline with a one per cent. solution of sodium carbonate, or with the decinormal alkaline solution and about two c.c. of a one per cent. solution of calcium chlorid are added to it. This is next mixed with an equal quantity of sweet milk, and placed in the thermostat. The alkalinization has destroyed the ferment, and the calcium chlorid will convert the labzymogen, if present, into labferment, and the coagulation will take place in the usual time.

The Quantitative Tests.—The quantitative estimation of the labsecretion is roughly done by dilution, an excellent clinical method, given by Boas. If the qualitative test for the labferment has been positive, we exactly neutralize the filtered contents, and make four dilutions with distilled water: 1 : 10, 1 : 20, 1 : 30, and 1 : 40. To five c.c. of each of the dilutions we add five c.c. of milk, place all in the thermostat, and note the weakest dilution in which the coagulation occurs.

For the quantitative estimation of the labzymogen, the neutralized contents are made slightly alkaline, and dilutions prepared of 1 : 10, 1 : 20, 1 : 40, 1 : 80, and 1 : 160. To five c.c. of each add two c.c. of the one per cent. calcium chlorid

solution, and five c.c. of milk, and place all in the thermostat, and mark the weakest dilution in which the coagulation occurs.

Normally, the end dilutions 1 : 40 (labferment) and 1 : 160 (labzymogen) should give a positive result.

If the gastric contents contain no free acid, before basing a conclusion on a negative result we should introduce—and withdraw half an hour later—a glass of $\frac{1}{20}$ normal HCl in the morning, when the stomach is empty. When there is no free hydrochloric acid in the contents, this method is absolutely necessary, in order to confirm or control a negative result after a test-meal.

If the gastric contents be neutral, labferment is absent, but labzymogen may be present even in normal quantity.

The Practical Value of the Labferment Signs.—Practically, we may find three conditions: the labsecretion may be normal, variable, or persistently diminished.

1. The constant presence, after a test-breakfast, of the labferment and its mother substance in normal quantity, does not always exclude an anatomical disease. But in a very large majority of cases this sign speaks distinctly in favor of a dynamic affection, and this rule is without exception when the acid secretion and the motor function are also normal. But in continuous excessive HCl secretion, due to glandular gastritis, the test-meal contents show usually an increase of labsecretion, and labferment is often present in greater quantity than labzymogen. In gastritis, associated with excessive HCl secretion, the labferment is above normal, although the quantity of labzymogen in the contents removed at the end of one hour may be diminished. If the contents be removed sooner, the quantity of mother substance will be found at least equal to that of health, and the quantity of the converted ferment is usually normal or above normal throughout the evolution of digestion. The rule which claims that a normal milk-curdling power of the gastric contents excludes all but the dynamic affections does not obtain in many cases of glandular gastritis, of ulcer, and of complicated myasthenia.

2. A variable labsecretion is a common sign of incipient and mixed forms of gastritis, or of myasthenia and obstructive stagnation or retention. The quantity varies because the interstitial inflammation varies, and because the evacuation of the contents of the stomach is irregular.

3. The labsecretion may be persistently diminished. If this sign be established—and our conclusions should not rest on a single examination—there is glandular disease, and the

degree of deficiency indicates the degree and diffusion of the gastritis, or it indicates glandular degeneration. Consequently, the functional sign may make the exact diagnosis indicate the prognosis, and dominate the treatment.

(*b*) **Pepsin and Pepsinogen.**—Pepsinogen, the mother substance of pepsin, is formed continuously and, in all probability, by the chief cells of the gastric glands, and is stored in these cells to be poured out under the influence of digestive stimulation. The chief cells are at once the factory and the storehouse of pepsinogen, which, when given out as one of the elements of the specific secretion, comes in contact with the hydrochloric acid or the secreted chlorids, and is rapidly converted into the active pepsin. The quantity of pepsinogen converted is conditioned by the percentage of free hydrochloric acid, about 2.5 parts a 1000 being the most favorable strength. The organic acids also possess this power of conversion.

In the contents after a test-meal, the presence of pepsin is demonstrated by a positive biuret reaction. But the existence of pepsin-hydrochloric acid products is no proof of the secretion of pepsinogen in sufficient quantity, although the sufficiency of this secretion is suggested by the disintegration and solution of the bread and meat; but acid and water may accomplish this solution without the aid of pepsin. Consequently, not only when there is no free HCl in the contents, but also when this acid is present in normal or in excessive quantity, the quantitative estimation of pepsin should be made by testing the power of the properly acidulated filtrate to convert the normal quantity of albumin into albumoses within the proper time. The following methods have been used:

Method of Schiff (1868).—Brücke (1859) added to the gastric contents a small piece of hard-boiled white of egg, or of fibrin, and made the digestive power proportionate to the rapidity of the solution of the fibrin or the white of egg. Schiff estimated the quantity of albumin or fibrin dissolved in Brücke's experiment, after the lapse of a certain interval, by the increase in specific gravity. The specific gravity of the fluid before and after the artificial digestion is taken, and from the difference the quantity of substance dissolved is aclculated.

Method of Grützner (1874).—Grützner stained a mass of fibrin with ammonia-carmin for twenty-four hours, washed thoroughly with water, and poured over it a 0.2 per cent. solution of HCl. One-half gm. of the stained fibrin, in flakes, is

placed in the digestive solution, and the coloration, after various intervals, is compared with a standard carmin scale.

Method of Leube.—Two tests are made, the one with the gastric contents alone, and the other after the addition of pepsin. If the latter dissolves an equal quantity of albumin more rapidly than the former, the pepsin is deficient. Boas compares the rapidity of the solution of albumin by the gastric contents to be tested with the rapidity of the solution of the same quantity of albumin by the normal contents.

Method of Jaworski (1887).—Twenty-five c.c. of the clear or filtered gastric contents are divided into two equal parts, which are placed in separate glasses. To one, a drop of concentrated officinal HCl is added. A piece of hard-boiled white of egg (1.5 mm. thick, ten mm. in diameter, and weighing six centigrams) is placed in each glass. The two preparations are put into the thermostat at 40° C., the time is noted when the egg cylinders are dissolved, and the solution is then tested for peptone. Three c.c. of a five per cent. solution of KOH are added, and into the strongly alkaline solution a one per cent. solution of $CuSO_4$ is let fall, drop by drop, out of the buret. The solution of copper is added until the reddish coloration is no longer increased thereby. The greater the quantity of peptone, the later the peptone reaction begins and the later it reaches its greatest intensity. The quantity of peptone present is indicated by the intensity of the color: Hardly perceptible pale rose, mere trace; rose, trace; reddish, plain trace; red, moderate quantity; and dark red, large quantity. Syntonin and propeptones do not interfere with the reaction.

If, after twenty-four hours, the egg cylinders have not disappeared and the peptone reaction is negative, the test specimen possesses no digestive power. Normally acidulated specimens of the gastric contents, obtained at the height of gastric digestion, completely dissolve the egg cylinder within one hour.

Method of Hammerschlag (1894).—Three gm. of commercial egg-albumin are dissolved in 150 c.c. of a solution containing four parts of HCl per 1000. The solution, after standing twenty-four hours, is filtered, and the filtrate contains about one per cent. of albumin. With this preparation three tests are made.

Ten c.c. of the acid albumin solution are mixed with five c.c. of the gastric contents, and an Esbach tube, *a*, is filled to the mark U with the mixture.

To another ten c.c. of the preparation five c.c. of distilled

water are added, and with this a second Esbach tube, *b*, is filled to the mark U.

A third tube, *c*, is filled to the mark U with a mixture consisting of ten c.c. acid albumin solution, five c.c. gastric contents, and 0.5 gm. pepsin.

All the tubes are placed in the thermostat at 37° C. for one hour, removed, and filled to the mark R with Esbach's reagent.

After standing twenty-four hours the quantity of sediment is read, and the diminution of the sediment in the tubes *a* and *c* is compared with the sediment in the tube *b*. The peptonization is represented by the unprecipitated albumin products. If, for example, tube *a* contains three per cent. precipitated albumin and the control tube *b* contains six per cent., the digestive work is 50 per cent.

Method of Klug (1895).—Tube peptonization, according to this writer, is most active in a preparation containing 0.5 per cent. to 0.6 per cent. free HCl and 0.1 per cent. pepsin. A greater or less percentage than 0.1 per cent. pepsin diminishes peptonization.

The test may be made in the following manner: Twenty-five c.c. of the filtered contents, in order to eliminate the influence of the hydrochloric acidity, are brought to a free HCl acidity of 0.5 per cent. To this acidulated specimen are added ten gm. of finely divided hard-boiled white of egg, and the mixture is placed in the thermostat at 40° C. for twenty-four hours. After this period no more of the egg is utilized, and the further process consists in the transformation of the syntonin and albumoses into peptones.

For the differential estimation of the quantity of syntonin, albumoses, and peptones in the test mixture, Klug uses the biuret reaction, the intensity of which is measured by the spectrophotometer of Glan. The color extinction coefficient is obtained in the usual manner, that part of the spectrum between D 75 E and D 100 E being used.

The digestive mixture is boiled and filtered; the filtrate contains all the utilized albumin. To ten c.c. of this filtrate add five c.c. of a concentrated solution of caustic soda and six drops of a ten per cent. solution of cupric sulphate; shake and carefully filter to remove the excess of $CuSO_4$. The coefficient is then taken (E).

The remainder of the filtrate is exactly neutralized with a solution of caustic soda and filtered. The syntonin is thus removed: Ten c.c. of the filtrate are treated as before and the coefficient taken (E^2). Ten c.c. of the syntonin-free filtrate are next boiled with an excess of pure sulphate of ammonium

and filtered after cooling. The albumoses, except possibly deutero-albumose, are thus removed: To one c.c. of the filtrate free from albumoses are added five c.c. of concentrated solution of NaOH and three drops of the ten per cent. solution of $CuSO_4$. Filter repeatedly before taking the coefficient (E^3). $E^2 - E^3$ = albumoses. $E^1 - E^2$ = syntonin. E^3 = peptones.

Method of Oppler (1896).—Oppler's method is correct in principle, but it is long and tedious in practice. It is the best research method, but the method of Hammerschlag is sufficiently accurate for clinical purposes.

A neutral two per cent. solution of egg-albumin, preserved by the addition of 5 : 1000 chloroform, is mixed in definite proportion with a dilution of the gastric filtrate of a definite HCl (77, or 0.281 per cent.) acidity and placed in the thermostat for three hours. The quantity of nitrogen—and consequently of albumin—is estimated in the solution of dried egg-albumin, in the diluted contents filtrate, and in the mixture (placed three hours in the thermostat at 37.5° C.) after the removal of the undigested albumin. According to Oppler's investigations, 50 c.c. of the total (one hour after the test-breakfast) gastric contents, after dilution to the following quantities, digests during three hours the following percentages of the albumin in 20 c.c. of the two per cent. solution of commercial egg-albumin.

When	diluted	to	1	liter,	about	70	per	cent.
"	"	"	2	"	"	67	"	"
"	"	"	5	"	"	60	"	"
"	"	"	10	"	"	50	"	"
"	"	"	20	"	"	45	"	"

A 1 : 1000 solution of pepsin (pepsin, one; acid hydrochloric, 20; aq., 1000) digests in three hours about 35 per cent. of the albumin in the solution.

The test of digestive power is made in the following manner: The Ewald-Boas test-breakfast is given on an empty stomach, and one hour later the contents are removed; the stomach is washed with repeated small quantities of distilled water until the return fluid is clear and the mixed wash-water and expressed contents are diluted to one liter (or two) and brought to an acidity of 77 (0.281 per cent.) by the addition of dilute HCl. Fifty c.c. of this acidulated dilution are mixed with 20 c.c. of the two per cent. solution of egg-albumin, and the mixture is placed in the thermostat at 37.5° C. for three hours. The mixture is removed from the thermostat, exactly neutralized by the addition (quantity necessary being pre-

viously determined) of NaOH, boiled, acidified with acetic acid, five c.c. saturated solution of NaCl are added, and the mixture is again boiled, washed with enough distilled water to make 150 c.c., and filtered after it has become cool. (Oppler makes a control test at the same time.) The quantity of nitrogen is estimated in 50 c.c. of the filtrate by the Kjeldal method, and the result is multiplied by three. The quantity of nitrogen in the dilution of the contents—and in the H_2SO_4 used as a reagent—is deducted. The remainder is the quantity of digested albumin. If, for example, the solution of egg-albumin contains 2.1 per cent. albumin, and 50 c.c. of the diluted (one liter) total gastric contents contain 77.5 milligrams of N, and the 50 c.c. of the test digestion filtrate contain 22.4 milligrams of N, then 22.4 × 3, or 67.2 milligrams of N have been digested. Of this quantity 17.5 milligrams are already present in the gastric contents. Consequently, the remainder, after this is subtracted, when multiplied by 5 (20 c.c. being used), is equal to the quantity of the albumin in 100 c.c. of the test albumin solution which has been digested. The 248.5 milligrams of N are equal to 1.55 gr. of albumin, which is 74 per cent. of 2.1. That is, the digestive power is a little greater than normal (70 per cent.), or a little more than twice as great as the digestive power (35 per cent.) of the 1 : 1000 solution of pepsin.

The Practical Value of the Pepsin Signs.—Pepsin may be formed in normal, in excessive, or in subnormal quantity. A continuous normal pepsinogen secretion is a good sign of the integrity of the glandular layer; but a mild anatomical disease of the stomach may be present without causing a noteworthy change in the quantity of pepsin.

Pepsin, contrary to the common belief, may be secreted in excessive quantity. This excessive secretion is often met with in chronic hypersthenic (glandular) gastritis. The early morning contents of continuous secretion often digest more rapidly than the contents obtained after a test-meal given to the same individual. The fluid of gastric retention with free HCl usually digests better when diluted and acidulated with HCl. But if the irritation and continuous secretion and the accumulation of ferments be controlled for a few days by diet and lavage, the actual diminished power of secretion may be made clear in some cases of gastric retention. Consequently an excess of pepsin may be due to the secretion of a gastric juice which is excessively rich in it or to its accumulation in the stomach. In both instances the tests of Hammerschlag and Oppler give an increase of digestive power,

and the dilutions likewise digest more albumin than do similar dilutions of the test-breakfast contents. Whenever the filtrate of the early morning contents possesses greater digestive power than the filtrate of the test-breakfast contents, there is motor insufficiency. In simple continuous secretion, the early morning contents possess no greater digestive power than the contents after the test-breakfast, and the specific gravity is that of the gastric juice—1004 to 1006.

In still another condition the pepsin tests are valuable, as when there is persistent and progressive diminution of the specific elements of secretion. This is a physical sign of chronic asthenic gastritis, of atrophy of the gastric glands, or of carcinoma. But pepsin secretion is commonly diminished in chronic inanition, and variations of quantity occur in consequence of nervous influences, particularly in hysteria and in adenasthenia gastrica. Consequently, not even a great diminution of the secretion of this ferment should be considered pathognomonic of severe glandular disease without other corroborative signs. The diminution of pepsin is not characteristic of any particular disease of the stomach, and its quantity varies in very close relation with the quantity of total HCl. Consequently, its increase or decrease or its presence in normal quantity in the contents after the test-breakfast possesses about the same significance as like states of HCl secretion. But there is no doubt that the diminution and the loss of labferment secretion are very grave signs and are much less frequent than the diminution and loss of hydrochloric acid and pepsin secretion.

3. Mucus, or the General Secretion.

The general secretion of the stomach is mucus, a product of the cylindrical cells which thickly line the surface and extend a short distance into the peptic glands and line completely the mucus glands. This secretion forms a very important protection to the delicate structures which it normally covers as a thin layer. In catarrh, the quantity of the mucus may be greatly increased, and forms, particularly about the pylorus, very thick masses, either clear and tough with a pale membrane beneath or tinged with blood and mixed with the exudate from the hyperemic blood-vessels.

The mucus which is secreted by the normal cylindrical surface epithelium contains only a trace of mucin, and no cloudiness is produced by the addition to it of either distilled water (dilution) or acetic acid. It is readily dissolved and

digested by the gastric juice. Consequently, the chemical test of normal gastric mucus is worthless, and the search for excessive mucus secretion should be made, not in the test-meal contents, but in the early morning wash-water. In the morning before breakfast ½ of a pint of water is allowed to flow in and out of the stomach (siphonage) several times, and this wash-water is examined for mucus. The stomach mucus will then appear in shreds and in flocculent masses mixed with a few fat droplets, starch granules, and cylindrical epithelial cells. It stains but faintly with methyl-green and thionin, and it swells, instead of coagulating and contracting, on the addition of acetic acid. The gastric mucus, however, which is formed after the transformation of the cylindrical into goblet cells (gastritis), contains more mucin, and consequently stains more intensely and precipitates on the addition of acetic acid. The presence of much mucus in the wash-water or in the stomach-contents delays filtration, and large quantities of it may be left on the filter. The greatest quantities of mucus are found in asthenic and atrophic gastritis and in carcinoma. It accumulates because it is secreted in excess, contains more mucin, and is not dissolved by digestion. The quantity of undissolved mucus is in inverse proportion to the quantity of HCl and pepsin. The persistent secretion of mucus in excess is a distinctive sign of gastritis—be the gastritis primary or secondary, acute or chronic, asthenic or hypersthenic, or atrophic.

The mucus removed from the stomach may have been swallowed. The swallowed mucus forms glairy lumps, mixed with squamous epithelium and often with pus cells, and it frequently floats on the surface. The stomach mucus occurs in shreds or flocculent masses, is mixed with starch granules, contains cylinder or beaker cells or their nuclei, and only a few leukocytes. The collection of mucus in the stomach may in reality be an accumulation of saliva. The saliva may be detected by the reaction of the sulphocyanid of potassium which it contains with iron. A dilute solution of chlorid of iron is added, drop by drop, until the red color which is produced no longer increases in intensity. The coloration remains after the addition of hydrochloric acid and is not discharged by bichlorid of mercury, otherwise the red color is not produced by saliva.

2. THE MOTOR FUNCTION.

The gastric muscle plays an exceedingly important part in the pathology of the stomach. Motor insufficiency is a serious primary trouble, and likewise a serious complication. The cardiac muscle has recently been given its proper place in the pathology of the heart. For a long period attention was directed chiefly to the valves and to the pericardium; the heart muscle was neglected. But the involuntary muscular system deserves a more prominent place in internal pathology. Attention has been directed too exclusively to the mucous membrane. But the uterine muscle is no more important in labor, nor the heart muscle in the circulation of the blood, than is the gastric muscle in digestion. The integrity of the muscle cells is no less important than that of the cells which secrete.

When the general strength and nutrition are affected in an unfavorable manner by a disease of the stomach, this result can usually be attributed to a motor defect, which, unlike secretory insufficiency, can not be compensated. The chemical work of the stomach may be null without affecting nutrition if the integrity of the motor function is maintained and the contents of the stomach are given over to the healthy intestines for digestion and assimilation. The intestinal juices are much more powerful and active than the gastric secretion. This is the teaching of operations on the stomach which simply secure the passage of the food into the intestines; such is also the teaching of experiments on animals and of pathology.

The movements of the stomach are two—the evacuating and the churning. The movements are in all probability due to the excitation of the ganglia in its walls, through which the vagosympathetic branches which go to the stomach probably also exert their influence. The stimulant of these movements is not HCl only; the movements continue when the reaction is neutral, but are excited by the various elements of the contents.

The movements of the stomach during digestion and the process by which the organ empties itself have long been the subject of careful study. The character of these movements is no less a matter of controversy than is their explanation. The philosophy of the subject may be left out of account in a clinical work and only the results of observations need be gathered.

Viewed from the results, the contraction of the muscular layer of the stomach produces a twofold effect—the increase of intragastric pressure and the motion of the contents. The fibers the contraction of which causes these results may act contemporaneously and in union or separately and independently.

During the first period of digestion, the duration of which is dependent upon the physiological action of the food, the tonic contraction predominates, and the motionless gastric wall applies itself closely to the gastric contents. This period of high intragastric pressure may last a few minutes or two or three hours, the duration being determined by the physical and chemical qualities and the physiological action of the contents and by the power of those fibers which have to do with the result.

The second period is that of the worm-like movements, which are cardiac, pyloric, or total, according to the location and extent of their visible expression.

The movements of the cardiac portion begin at the cardia, are slow and weak, and lose themselves in the middle of the organ. These peristaltic movements may be contemporaneous with those of the pylorus or they may be alternating. Observation has established between them no law.

The movements of the pyloric end are more complex. Two forms have been observed and may be accepted as physiological: First, those which begin on the cardiac side of the pylorus and become stronger as they pass forward and disappear in the duodenum; second, those which begin on the descending portion of the duodenum and move backward on to the stomach, in the middle third of which they are lost, soon to reappear with greater power along the pylorus to the duodenum. The character of these movements forms the basis of a theory which maintains that the opening of the pylorus is due to a cause which has its origin in the duodenum.

The total movements are exclusively peristaltic, and begin at the cardia and move slowly toward the pylorus, near which the wave becomes higher and more rapid. The effect of the peristalsis is more visible along the greater curvature, the thick longitudinal bundle along the lesser curvature serving as the fixed line of attachment. These lesser curvature longitudinal fibers extend to the duodenum, over the upper and anterior surfaces of which they spread. Their contraction would tend to straighten the line between the cardia and the more immovable duodenum, and, if not counteracted by contraction of the ring fibers, it would open the pylorus.

The excitation of the splanchnic in the thorax, according to Oser, opens the pylorus and stops its rhythmic contractions. Oppenheimer explains this result by the contractions of the intestinal arterioles and by intestinal contraction on account of the consequent anemia. Such an anemic condition is present when the intestines are empty and at rest. The intestines become shortened in inanition, and all conditions that increase absorption and nutritive change also cause the stomach to empty itself more rapidly. Such is likewise the effect of purgatives which act on the intestines. This would suggest the possibility of the opening of the pylorus through the contraction of the longitudinal fibers of the duodenum extending on to the stomach.

The movements of the stomach are excited and controlled by the ganglia in its walls; these ganglia are brought into activity by the properties of the gastric contents and by the impression received through the vagosympathetic nerves. Particular centers or areas in the central nervous system are connected with the gastric muscle. The vomiting center is the same as that of respiration (Grimm); or, according to Thomas, is a single center in the posterior part of the medulla oblongata, beginning two mm. before and ending three mm. behind the calamus scriptorius. The centers for the contraction of the cardia are located in the corpora quadrigemina, the fibers from which run partly in the cord (to the eighth cervical vertebra, and thence to the stomach in the splanchnics) and partly in the vagi: the centers for the movements of the wall are in the same bodies, but the fibers run exclusively in the cord and the thoracic sympathetic (Openchowski, Hlasko). The dilator centers of the cardia are situated near the anterior commissure in the vicinity of the union of the nucleus caudatus and nucleus lentiformis: the fibers from these centers run in the cord to the fifth cervical vertebra and also in the vagi (Knaut) on their way to the cardia.

The normal motor function is dependent on a proper nerve supply, on a healthy muscle, and on a physiological excitation. Consequently, the motor function may be deranged through the central and sympathetic nerves, by a badly nourished, weak, or diseased gastric muscle, and by improper contents in the stomach. The muscle may be hindered in its work by adhesions, deformities, displacements, and obstruction to the evacuation of the stomach. Several methods have been given for testing the motor function.

Some valuable information concerning the motor function may be obtained from the quantity of the contents after a

meal given to test the secretory activity of the stomach. Normally, the total quantity of the contents of the stomach after the test-breakfast is about 125 c.c.; after the test-meal of Sée, about 175 c.c.; and after the test-dinner of Riegel, about 200 c.c. If these values are increased more than 25 c.c., there is motor insufficiency; or if they are decreased more than 25 c.c., the evacuation of the stomach is too rapid—provided the increase or decrease is not produced by supersecretion or subsecretion respectively. The total quantity of the contents must be estimated by the method of Mathieu and Rémond or by the method of Strauss.

The method of Mathieu and Rémond is based on the principle that the acidity of the contents before removal from the stomach is equally diffused. Enough of the contents is first removed by expression for analysis, and the vessel containing it is set aside. Two hundred c.c. of distilled water (or a particular quantity) are next introduced into the stomach through the tube and well mixed with the contents by alternately lowering and raising the funnel so as to allow the mixture to flow back and forth between the stomach and the funnel. The diluted contents are then removed by expression and received in a special vessel. The quantity of the undiluted contents (p) and its total acidity are known. The quantity of water used (w) and the acidity of the diluted contents (a^1) are also known. Hence: $ax = a^1w + a^1x$ $\therefore x = \frac{a^1w}{a-a^1}$. The total contents are consequently equal to $p + \frac{a^1w}{a-a^1}$. This method, commonly ascribed to Mathieu and Rémond (1890), was first published by Jaworski (1882). Strauss estimates the total quantity from the specific gravities of the diluted and undiluted contents. If x represents the quantity left in the stomach after the first expression, s and s^1 the specific gravity of the undiluted and diluted contents respectively, and w the quantity of distilled water used, $x = \frac{w(s^1-1)}{s-s^1}$. The total quantity is consequently equal to $p + \frac{w(s^1-1)}{s-s^1}$. Goldschmidt employs only 50 c.c. of distilled water for diluting the unexpressed contents, and adds distilled water to the undiluted contents until the specific gravity is reduced to that of the diluted contents. The quantity of distilled water which is required for this purpose represents the quantity of contents left in the stomach after the first expression, which should be as complete as possible.

The bacteriological as well as the functional signs give some information concerning the motor work done by the stomach, as stagnation and retention are two of the conditions favorable to germ development. But methods have been devised for testing the motor function more directly. Before describing these direct methods the ingenious method of Ewald and the old plan of Chomel will be discussed.

Method of Ewald and Sievers (1887).—The salol test of Ewald, as this great clinician himself admits, is only a very crude one. Salol is insoluble in the normal gastric juice, and is decomposed in the intestine into carbolic and salicylic acids. The latter undergoes absorption, and is eliminated as salicyluric acid in the urine. If about 15 grs. of the salol be given in capsules at the height of digestion by the normal stomach, the salicyluric acid may be detected in the urine in about sixty minutes, sometimes in thirty minutes, and sometimes its elimination begins in seventy-five minutes. The longer the lapse of time before its appearance in the urine, the greater is the motor insufficiency.

The test for the salicyluric acid in the urine is a very simple one. A few drops of the urine are allowed to fall on a piece of filter paper, and three or four drops of a ten per cent. solution of neutral chlorid of iron are placed so near the edge of the wet spot as to mingle with it. The violet color formed reveals the presence of salicyluric acid. A mere trace may be detected by first strongly acidulating a portion of the urine with HCl and testing, with the chlorid of iron, the residue of the evaporation of an ether extract.

Huber claims that the time of disappearance of the salicylic acid from the urine is a better measure of the motor insufficiency than the moment when it can be first detected. In health, the salicylic acid should all be eliminated in twenty-seven hours, and the persistence of the reaction beyond this period is proportionate to the motor insufficiency. This brings in the very uncertain factors of absorption, elimination, and the rapidity of the decomposition of the compound; and clinically the liability to error is greater than in the simpler test of Ewald.

The saliva decomposes salol, and this may occur in the stomach if it contain much alkaline mucus or exudate. Bacteria decompose it in the stomach. The myasthenic or incompletely obstructed stomach does not empty itself all at once, and at a particular moment, but intermittently throughout the period of digestion, either too slowly (stagnation) or never completely (retention). These facts would suggest the

fallacies of the method. But theoretical considerations are of little value when the practical test can be made. The application of the method at the bedside reveals the very limited value of the test and the inconstancy of the results. The necessity of urinating every ten minutes until the salicyluric acid appears in the urine renders the method inconvenient for men and impracticable for women. Fleischer gives a grain and a half of iodoform at the beginning of a meal, and notes the moment of the appearance of iodin in the saliva. The information obtained through the salol and iodoform tests is comparatively insignificant.

Method of Chomel.—The splashing sounds give some information concerning the state of the gastric muscle during digestion, and concerning the time which the stomach requires for the complete evacuation of its contents. A strong and healthy stomach splashes but little or intermittently, or it does not splash at all during the digestion of an ordinary meal. Splashing throughout the period of digestion and which is demonstrable from day to day, is a sign that the stomach muscle does not properly contract on its contents and perform its churning work. This test of the tonicity of the muscle may be made during digestion, or after the administration of a glass of cold water during the period of functional repose. The splashing sounds are also used to test the evacuating power of the stomach. Splashing can not be produced when the stomach is empty, and it always denotes that the stomach contains fluid and gas. If splashing can be elicited when the stomach should contain none of the previous meal, the evacuation of the stomach is delayed. If it can be produced just before the second and third meals of the day, stagnation of the second degree is present. If the splashing can be produced in the morning before breakfast, it is a sign of retention. The validity of these signs depends on two conditions—namely, no fluid must be swallowed during the period between the end of the meal and the examination, and excessive or prolonged or continuous secretion must be excluded. The method of Chomel is valuable as a rough preliminary test.

Method of Leube.—The method of Leube, with the modification of Boas, is a most excellent and complete test of the evacuation sufficiency of the stomach. The patient is directed to eat a plate of soup, one roll, and a beefsteak. Seven hours later the stomach should normally be empty. If the stomach is washed out at this time, and a notable residue of the meal is obtained, the stomach has failed to evacuate itself within

the proper period. This failure may be due to myasthenia or to obstruction, or to both, or to excessive secretion. Leube's method establishes an evacuation insufficiency, but not the degree of it.

To determine the degree of motor insufficiency, Boas recommends that an evening meal consisting of two cups of tea, two small rolls, and a cold meat be given at 8 o'clock, after thorough lavage, unless the stomach is already empty and clean. No visible (macroscopic) remnants of this meal should be in the stomach on the following morning before breakfast, unless motor insufficiency of the second degree (Boas) exists. Naturally, other meals than those of Leube and Boas may be given; but whatever meal is prescribed, the moment of its complete evacuation by the normal stomach should be known, and used as the measure of motor sufficiency.

In practice it is best to learn how the stomach conducts itself with reference to the usual meals—breakfast, lunch, and dinner, or breakfast, dinner, and supper; for in dietetics it is categorically imperative that the stomach should be empty at the beginning of each meal, and we accordingly recognize three degrees of motor insufficiency: In mild stagnation the evacuation of the stomach is delayed, but the stomach empties itself completely between each meal; in severe stagnation the stomach is found empty only in the early morning before breakfast; in retention the stomach is not empty at any moment during the twenty-four hours, and in the morning before breakfast it contains food and digestive products, the contents being more acid than the contents removed at the acme of the digestion of a test-meal, and of a higher specific gravity than the gastric juice (1004 to 1006). If the stomach be found empty before each meal, and if the evacuation of the stomach be delayed, as revealed by abnormal splashing, or by too large a quantity of contents after the test-meals, or by the presence of contents at the moment after a particular meal when the stomach should be empty, there is mild stagnation or excessive secretion, or possibly the two conditions coexist. If the stomach be empty only in the morning before breakfast, there is severe stagnation or prolonged digestion due to excessive secretion, or the two conditions may coexist. If the stomach is not empty before breakfast, there is retention or continuous secretion, or both conditions may be present. The differentiation of excessive and continuous secretion from myasthenic and obstructive stagnation and retention is discussed in Sections IV and V.

But how can it be determined that the stomach is empty before meals or at a particular moment after meals? If the stomach splashes it is not empty; if contents can be expressed or aspirated, it is not empty; but when no splashing can be elicited and when no contents can be expressed or aspirated the stomach may not be empty. If the tube be introduced into the stomach until its tip touches the greater curvature, and if air be forced through the tube by compressing an attached bulb, the ear, applied over the stomach, will detect bubbling when the stomach is not empty. Or if 100 c.c. of a one per cent. solution of sugar be introduced into the stomach, the percentage of sugar will be diminished through dilution by the contents. The exact quantity of the contents can be estimated by dividing 100 by the percentage of sugar in the expressed or aspirated dilution (determined by the ammonia and copper mixture) and subtracting 100 from the result.

Method of Mathieu.—The method of Mathieu and Hallot is an excellent one. It is a very great modification of the old oil method of Klemperer. A test-meal containing a certain quantity of emulsionized oil is given, the tube is introduced after a stated interval, and the contents are removed. The entire quantity of oil in the removed contents is estimated. The part of the oil evacuated represents the motor work.

The test-breakfast, which is eaten in the morning on an empty stomach, has the following composition: 60 gm. of bread; an emulsion of 10 gm. of oil of sweet almonds, 5 gm. of powdered gum arabic, 30 gm. of simple syrup, and enough weak tea to make 250 cm. One hour after the beginning of the meal the contents are removed and the total contents estimated by his dilution (200 cm.) and acidity method. Twenty-five cm. of the undiluted contents are finely divided in a mortar, neutralized with NaOH solution, mixed with sand, and evaporated on a water-bath. The oil is extracted with ether, the ether is driven off by evaporation, and the quantity of oil is weighed. The total quantity of oil is easily calculated, the total quantity of the contents being known. Normally, at the end of one hour about four gm. of the ten gm. of oil are recovered.

The author of this method uses it also as a secretion test, and saves a part of the undiluted contents for chemical analysis. From the data he also calculates the part of the removed total contents which consists of saliva and gastric secretions, and the part which consists of the administered test-meal. Suppose, for example, after one hour four gm. of

oil are recovered. Four-tenths, or 100 cm., of the test-meal, consequently, were left in the stomach. This quantity, subtracted from the total quantity of contents, gives the portion of the contents which consists of gastric secretion and swallowed saliva.

The Water-test.—None of these motor tests give the cause of the motor insufficiency. The stagnation or retention may be due either to obstruction or to myasthenia. The stagnation of myasthenia is a stagnation of liquids. In obstruction (incomplete), liquids are evacuated much more readily than semi-solid food. The normal stomach evacuates 500 cm. of water (just cold enough to be refreshing) in one to one and one-half hours. If the tube introduced after the expiration of one and one-half hours recovers a notable quantity of water there is motor insufficiency which is in all probability due to myasthenia, unless obstruction is so great as to cause almost complete retention. If food stagnates or is retained, and the water is evacuated within the normal period, the trouble is obstruction. If the quantity of the contents after a test-meal is abnormally large and the specific gravity below 1010, and if the water is evacuated within the normal period, there is excessive secretion, or excessive secretion with obstruction. The water-test is useful in detecting myasthenia (first and second degrees of stagnation) and in distinguishing obstruction from myasthenia during their stagnation stages.

Methods of Dehio and Rosenbach.—Some information concerning the elasticity of the stomach may be obtained by the methods of Dehio and Rosenbach.

The method of Dehio was intended for the purpose of locating the lower border of the stomach; but, as indicated by Boas, it may also give some information concerning the distensibility of the stomach. Normally, after one glass of water has been swallowed, the lower border of the dull area (the patient standing) is about 11 cm. below the tip of the ensiform process. Each succeeding glass carries the limit of the dulness about two cm. lower, until after the fourth glass the lower border is near the level of the umbilicus. If the glasses of water be given in succession, a more rapid descent of the dull area than normally occurs would indicate that the stomach wall yields too readily to pressure.

The method of Rosenbach is based on the same principle as the method of Dehio, but the change in the upper level of the water is determined by means of air blown through the introduced stomach-tube. One hundred c.c. of water are introduced into the empty stomach, and the distance of the level

of the fluid from the incisor teeth is determined by placing the ear over the stomach while air is slowly blown into the stomach through the tube by compressing an attached bulb. The cessation of the bubbling indicates that the eye of the tube (a tube with one opening should be employed) has emerged from the water. If a glass of water be introduced, the change in the level of the fluid is again determined. The less the rise in the level of the fluid, the greater is the distensibility of the stomach.

Some idea of the tonicity of the stomach wall may be obtained by attaching a manometer to the funnel end of the tube before its introduction into the stomach to withdraw the contents after a test-meal. The greater the intragastric pressure, the more strongly is the stomach contracted on its contents. The gastrograph of Einhorn and the apparatus of Hemmeter are of little clinical interest, although a practical method of accurately displaying the peristalsis of the stomach would be of very great value. One of the best practical indicators of the tonicity of the stomach is the difference in the size of the organ when it is moderately full and when it is empty. A stomach which does not retract when it is empty has lost its tone—its elasticity. The stomach retracts about the line of the fixed lesser curvature. Consequently, the change in the position of the greater curvature displays the change in the size of the organ produced by its retraction. Instrumental methods may be used to make known this change of position, but the physical signs ordinarily suffice for the purposes of practice.

The motor signs display either normal muscular activity and work or reveal some pathological variation of the motor function. The negative diagnostic value of the normal function should not be forgotten. When the functions are found to be right, as much information is obtained as when they are found to be wrong.

The pathological variations are excessive activity and insufficiency. Excessive activity is manifested by spasm—cardiospasm, gastrospasm, spasms of the pylorus, peristaltic unrest. Insufficiency is due either to a diminution of muscular power or to inability to overcome some mechanical interference—gastroplegia, myasthenia, displacement, inflammatory or cancerous infiltration of the muscular coat, pyloric or duodenal obstruction, perigastric adhesions, deformity of the stomach.

In regard to the evacuation of the stomach, it may be too rapid or too slow. Too rapid evacuation occurs in pyloric incontinence, which may be a dynamic affection, or a symptom

of an organic disease, particularly cancer (scirrhus), which has converted the pylorus into a rigid, open canal.

Delayed evacuation is most commonly the result of myasthenia, the forms and causes of which are discussed in the fourth section of this book. The other causes of delayed evacuation, when it is not a mere temporary condition or due to excessive secretion, are the anatomical diseases already mentioned, and which are described in the fifth section of this volume. It is important to remember that motor insufficiency may be absolute and relative, temporary and persistent, dynamic or anatomical, simple or complicated. The motor signs are all symptoms—not one of them is a disease.

3. ABSORPTION.

Little is known concerning the physiology and pathology of absorption by the human stomach. It is uncertain whether absorption, which occurs to a very small extent, is a physical process of diffusion or is the work of the cells which cover the mucous membrane. It is known that no water and fat are absorbed by the human stomach, but that alcohol, the sugars, and albumoses are absorbed in small quantity. In the experiments made on man in health and in disease, it has not been possible to tell how much the results obtained have been affected by gastric secretion and evacuation.

Jaworski (1883) determined the relative absorbability by the human stomach of a number of salts, acid carbonate of magnesia being the most absorbable. These results may be thus briefly presented: $H_2Mg\ (CO_3)_2 > HNaCO_3 > Na_2SO_4 > MgSO_4 > H_2NaPO_4 > KCl > FeCl_2 > NaCl$. These results may be incorrect for those salts which, like NaCl, exist in the gastric juice.

During the past two years (method and results described in a lecture at the Polyclinic in October, 1886) we have employed the following test of absorption: The yolks of four eggs are thoroughly beaten and mixed with 200 c.c. of distilled water, in which 25 gm. of dextrose (Merck) has been dissolved, and 30 c.c. of whisky are added. Two hundred c.c. of the mixture are given on an empty stomach, the balance being utilized for the estimation of the percentages of fat (ether extract) and dextrose (ammonia and copper solution). After one hour the contents are expressed and the total quantity of contents is estimated by the total acidity method of Mathieu.

The percentages of fat and dextrose in the contents are estimated and a qualitative test (liquor potassa and iodin solution) is made for alcohol. From these data it can be readily determined how much fat has been evacuated and consequently the proportion of the contents, how much of the contents consists of secretion, how the relative percentages (not affected by evacuation nor by dilution) of fat and dextrose have been altered, and consequently how much of the dextrose has been absorbed.

The old method of Penzoldt and Faber is of no clinical value. Three grains of pure iodid of potassium are administered in a capsule while the stomach is empty, and the moment of the appearance of iodin in the saliva is detected by using starch paper and fuming nitric acid. Normally, the reaction in the saliva is positive in fifteen minutes. In some diseases its appearance may be delayed; but these diseases (cancer, retention) are precisely the ones in which motor insufficiency occurs and free HCl is often absent. Little is known about the many circumstances which delay the absorption of the iodid, and the test has proved almost worthless at the bedside.

4. DIGESTIVE WORK.

The chemical transformation of the food, which is produced in the stomach by the saliva and by the gastric juices, represents the digestive work done in this organ. The digestive work which is performed in the stomach after a test-meal is displayed by the digestive products and by the portion of the meal that remains undigested. The digestive work is expended on the carbohydrates and on the proteids.

The carbohydrates are not digested by the gastric juice, but the transformation by the saliva is modified by the diseases of the stomach. The inspection of the expressed contents may show that the starch remains for the greater part unchanged, or has been normally digested, or has been rapidly and completely dissolved. The normal contents are a finely divided or nearly homogeneous mixture. If the starch digestion is arrested too early by excessive gastric secretion, the bread, or bread and potato, are only partly dissolved; Lugol's solution gives a blue or brownish coloration. If starch digestion is very active (subacidity), the solution and transformation is more complete; Lugol's solution gives

a reddish or no coloration, and Fehling's solution is positive. The accumulation of the products of starch digestion increases the specific gravity of the filtrate of the contents obtained after the test-meals, unless secretion is excessive. Normally, the specific gravity of the filtrate after the test-breakfast is 1010 to 1015; after Sée's meal, 1015; after Riegel's dinner, 1015 to 1020. If a large quantity of starch products is present, either the evacuation of the stomach is too slow or absorption is diminished, or both conditions are present. Digestive products do not accumulate in the normal stomach.

The digestion of the proteids is revealed by inspection and by chemical tests, and is displayed best by the Riegel or Sée meals. The proteids may be rapidly digested as in active or excessive secretion; or they may remain undissolved and untransformed in anacidity. The methods of distinguishing syntonin, albumoses, and peptones is described elsewhere, but the biuret test gives a rough idea of the activity of peptonization. The products of proteid digestion accumulate in the stomach only when there is motor insufficiency.

The digestive work done by the stomach in the various diseases is described in the fourth and fifth sections. By mere inspection of the physical properties of the contents, information concerning the functional power of the stomach may be obtained, which is very valuable both in diagnosis and in the dietetic treatment.

CHAPTER IV.

THE BACTERIOLOGICAL SIGNS.

It is a remarkable clinical fact that the micro-organisms of the stomach are quite constant, and are characteristic of the qualities of the contents and of the motor function of the stomach in which they grow. As a rule, germs do not thrive in an acid medium so well as in an alkaline or a nearly neutral culture. Consequently, a large number die rapidly or degenerate, or form the more resisting spores when they remain in the acid stomach. The germicidal power of the gastric juice is important, but it is incomplete, both in health and, to a greater degree, in disease. The germs swallowed

during the period of functional rest and during the period when no hydrochloric acid is free in the stomach, may escape uninjured into the intestines and find there a persistent and favorable soil. Their passage through the stomach may be too rapid to allow time for their destruction. The healthy stomach may fail to protect the organism against invasion even by the pathogenic germs. The diseased stomach may become a breeding receptacle, particularly for the saprophytic germs. The hydrochloric acid influences only the quality of the germ growth which occurs in the diseases of the stomach accompanied by motor insufficiency.

But apart from the acid reaction, the composition of the diet exercises a great influence on the development of the lower forms of life. Each germ has its own peculiar habitat, its favorite culture soil, and dies when it can not adapt itself to the sudden changes which occur in the contents of the stomach. On the other hand, their increase is rapid in a favorable soil.

But of more influence than either the acidity and the composition of the contents of the stomach are the intermittence of the food supply and the complete emptiness of the resting organ, which, when normal, evacuates the germs along with the chyme into the duodenum. Thus the normal stomach is intermittently empty and clean and without a culture soil. Consequently, in the normal organ germs do not have time, during the short digestive period, to manifest their very active powers of growth and proliferation. The churning movements of the stomach also help to keep its contents sweet. Constant motion is very destructive to some forms of germ life.

Naturally, the flora of the stomach is dependent upon the number of germs which obtain entrance there. The supply of these is abundant,—from food and drink, from the mouth, the nose, and the throat, and probably from the intestines. The opportunities afforded by pathological conditions are readily used.

The prevailing classification of bacteria is based on their form—cocci, bacilli, spirilli. Besides these, we have other germs—the molds and the yeasts. The particular kind of germ found, with but few exceptions, is of little diagnostic value. It is probable that a more complete study and a more exact control of the conditions would extend this short limit.

The quantity of germs found denotes more favorable conditions of growth. This may be referred to the quality of

the soil, but the richness and the active growth of the flora is, also, directly and closely dependent on the delay or the failure of the stomach to empty itself.

The germs of the stomach under consideration are not pathogenic, but live on and in dead matter. Consequently, their existence is made manifest by changes in the contents on which they live. The acids, the gases, and the toxins of fermentation and putrefaction are thus developed.

The bacteriological signs consist of the kind and the number of the micro-organisms, and of the products which they form by fermentation and putrefaction. The products which are of practical importance are the organic acids, the gases, and the toxins.

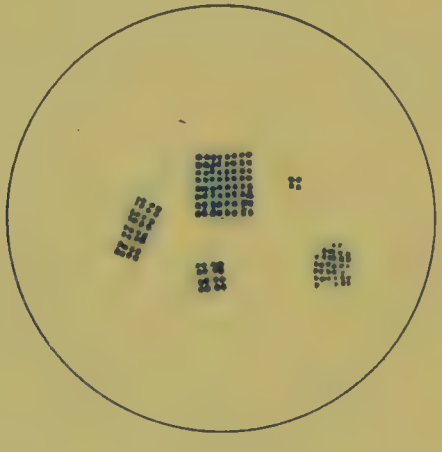


Fig. 10.—Sarcinæ ventriculi from stomach-contents, × 530; stained with methylene blue (authors' specimen).

1. The Kind of Germ.—The many kinds of germs found in the healthy stomach and in the pathological stomach have not been isolated and studied; but it will not be denied by those who frequently make a microscopical examination of the stomach-contents that the individual forms are very numerous. Only sarcina, yeast, and the bacillus geniculatus have a definite pathological meaning. And this is true of these three only when they are persistently present in large quantities and in active growth.

Many forms of sarcinæ exist in the air, and they may find their way into the stomach, and, under favorable conditions, may there proliferate. Oppler, who has best studied these cocci in the stomach-contents, succeeded in isolating five varieties, presenting distinct color and culture peculiarities. The cultures possess only a scientific interest; practically, we

are concerned only with their persistent presence in large quantities in the contents of the stomach.

These cocci are about 2.5 μ in diameter and appear in small cubical groups of eight, the packages or bales being marked by lines running at right angles. Larger packets may be formed. They may be found very loosely united or separated, and are small when undergoing degeneration.

Sarcinæ in large quantities are only found in benign forms of gastric stagnation or retention with free HCl. They can not live in the lactic acid contents of carcinoma. In small number they may be found in cancer, during the free HCl stage, in gastritis, in ulcer, in gastroptosis, and in the dynamic affections. Their presence in these diseases is inconstant and rare. Their persistent growth in large numbers is character-



Fig. 11.—Yeast from stomach-contents; × 530 (authors' specimen).

istic of retention due to myasthenia and to non-malignant obstruction.

Yeast, which grows by budding and occurs in single cells, or in strings of cells, clear and bright, and staining yellow with iodin, is often found in the stomach-contents; but it only grows and flourishes there when there are motor insufficiency and a suitable soil. The acidity of the contents of the stomach has little influence on the growth of the yeast. It matters little whether the reaction is alkaline, neutral, or strongly acid. Even excessive hydrochloric acidity does not arrest its growth, nor does carcinoma prevent its development. Whenever there is motor insufficiency yeast may be found, but it is not very vigorous except in gastric retention, when the yeast accumulates in large quantity, and the microscope shows that the plants are growing rapidly. The proliferation of the

yeast is proportionate to the motor insufficiency and to the richness of the diet in fermentable matter. Yeast is often present in small quantity in all the diseases of the stomach, except simple ulcer.

The **bacillus geniculatus** (Fig. 12) is present in very large numbers in carcinoma (Boas), and is sufficient to render a case suspicious. This bacillus consists of cells a little smaller than the bacillus subtilis, arranged often in a zigzag line, or in pairs, joined end to end so as to form an angle. It does not color with iodin, but colors homogeneously with fuchsin. It is large, devoid of motion, may be easily seen without staining, and is always present in carcinoma when the contents contain a notable quantity of lactic acid. It develops in acid-sweetened bouillon and produces lactic acid (Kaufmann).

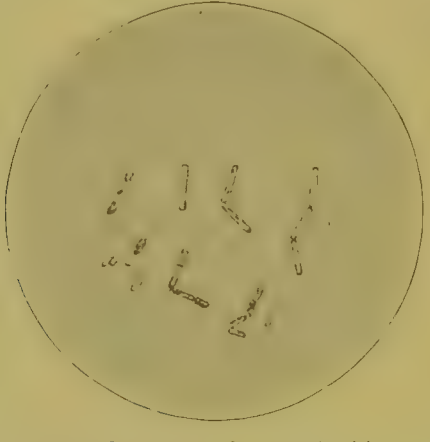


Fig. 12.—Bacillus geniculatus from stomach-contents; × 730: *a*, spore-formation; oval spores in center, ends pale and indistinct; *b*, multiplication by cell-division; *c*, normal cell arranged in a zigzag line; length, 4 to 8 μ; width, about 0.7 μ (authors' specimen).

These are the only varieties—viz.: sarcina ventriculi, yeast, and the bacillus geniculatus, whose simple growth in the stomach signifies that the organ is diseased. The sarcinæ, in large quantities, are almost exclusively found in benign retention with free HCl. Yeast finds a most favorable soil in stagnation or retention, regardless of the kind or the degree of acidity. The bacillus geniculatus is constantly present in cancerous obstruction of the pylorus which produces retention. It is sometimes absent in other forms of carcinoma, but its persistent presence in large numbers is almost characteristic of malignant disease of the stomach.

2. The Number of Germs.—The quantity and the general character of the micro-organisms present are of diagnostic value. The normal stomach is a bad medium for bacterial

growth, and the presence of germs here is seemingly tolerated only under the condition of quiescence; some forms are equal to this struggle for life, but are incapable of thriving. In motor insufficiency the conditions are more favorable, and the rapidity of development and the many varieties of germs are often remarkable; but possibly the many kinds may be only stages in the development of a few germs, and may be a sign of the rapid proliferation of a smaller number of distinct kinds than at first sight would appear to be the case. This lively pathological proliferation may be evident at a glance through the microscope and is a sign of gastric retention. Whenever the stomach completely evacuates its contents within the normal period, the number of germs found in it is never large.

3. Germ-Products.—The zymogenic and putrefactive bacteria are the most important in the pathology of the stomach. These, by their existence or growth, cause and perpetuate such changes in the chemical composition or constitution of the complex matter that the resulting substances become simpler and have a less force value.

This fermentative and putrefactive power is not without limitations, and the soil becomes exhausted and the process ceases. It is probable that all bacteria in the proper medium are zymogenic, and some are capable of producing both fermentation and putrefaction; but comparatively few possess these powers to such a degree as to form notable quantities of germ products in the stomach. Fermentation is much more common in the stomach than is putrefaction. The forms of fermentation of practical importance are lactic, butyric, acetic, and alcoholic fermentation.

(*a*) **Lactic Acid Fermentation.**—This form of fermentation is produced by a variety of germs, and is often followed by butyric acid fermentation, $C_6H_{12}O_6$ (glucose) $= 2(C_3H_6O_3)$ (lactic acid) $= C_4H_8O_2$ (butyric acid) $+ 2CO_2 + H_4$. The process is not so simple as represented by the equations, for other intermediate acids and gases are formed. Lactic acid may be destroyed by oxidizing germs.

The bacillus acidi lactici (Hueppe) is short,—about four times longer than it is thick,—motionless, builds spores, and is aërobic. It is very active at the temperature of the stomach, and converts glucose and lactose into lactic acid with the evolution of CO_2. It does not liquefy gelatin, and it forms colonies. Many other bacteria produce the same result. Among these are the bacillus geniculatus and two cocci found in the saliva. The bacterium coli commune, some-

times found in the stomach, is capable of producing lactic acid. Lactic acid may also be introduced into the stomach with the food and be separated from the lactates by the stronger HCl of secretion. The introduced and the liberated acid have no pathological significance.

Lactic acid possesses a definite diagnostic value. In many diseases of the stomach lactic acid may be formed in small quantities; but in few diseases is it produced in notable quantity, after the stomach has been thoroughly washed out in the evening and a lactic-acid-free test-meal has been given on the following morning. The formation of lactic acid takes place in the human stomach only in very special conditions, and these conditions are rarely fulfilled, except in carcinoma. In the first place, secretion must be diminished, and, indeed, to such an extent that no free HCl exists in the contents obtained at the usual time after a test-meal. The lactic acid bacilli are quickly rendered inactive by free hydrochloric acid, and even hydrochloric acid in organic combination suffices, when in notable quantity (0.12 per cent. or 35), to arrest lactic acid formation. Whenever lactic acid coexists with free hydrochloric acid, it has either been introduced into the stomach or formed in the stomach by the decomposition of lactates. In the second place, retention or malignant stagnation must exist. Even in achylia, lactic acid is not formed by germs in the stomach whenever the stomach completely evacuates its contents within the normal period. This rule is without exception, if the stomach be thoroughly washed out and a test-meal, like the oatmeal test of Boas, be given. The time such a meal remains in a stomach whose motor function is sufficient is too short for lactic acid to be formed by bacilli; but the motor insufficiency need only be slight, if it be associated with arrested peristalsis of a portion of the wall of the stomach. This localized arrested peristalsis may be due to adhesions or to a localized perigastritis, or to a new growth. Practically, however, the uncontrollable formation of a notable quantity of lactic acid in slight motor insufficiency occurs only in cancer, which permits the accumulation of germs and the retention of food upon its surface. Lactic acid fermentation may occur in either benign or malignant retention, accompanied by achylia. But the association of benign retention with achylia is very rare. Finally, the formation of lactic acid is dependent on the presence of lactic-acid-forming germs in a suitable culture soil. Any of the test-meals form a suitable soil, and the saliva may furnish the proper germs, which grow and accumulate in the stomach under special circumstances. The

activity of lactic acid formation is partly dependent on the richness of the contents in ptyalin digestive products.

The essential conditions of lactic acid formation make clear the diagnostic value of this bacteriological sign. Boas contends that the persistent uncontrollable formation of lactic acid in noteworthy quantity during the digestion of a saucer of lactic-acid-free oatmeal is a specific sign of cancer. This contention is almost universally true, the exceedingly rare exceptions occurring in diseases which usually possess other characteristic symptoms and signs, and which do not show the essential clinical characters of carcinoma.

Persistent, uncontrollable, and noteworthy formation of lactic acid may be an early sign of carcinoma. It is not a pathognomonic sign, but it is one of value, which should be confirmed by the presence of other symptoms of the same disease. It may not appear until late in the development of cancer. A malignant disease of the stomach may run its entire course without lactic acid formation. We have seen almost the entire gastric wall and the pylorus infiltrated, without the appearance of lactic acid in the repeatedly examined contents up to within a few days of death. The stomach, however, in this case was very small, and evacuated the test-breakfast completely in less than one hour. The absence of lactic acid formation does not exclude cancer. Its presence means that the conditions of its formation are realized. Its persistent formation in noteworthy quantity (0.05 to 0.2 per cent.) after thorough lavage, during the digestion of Boas' oatmeal test or of the Ewald-Boas test-breakfast, is an almost certain sign of carcinoma, for in achylia accompanied by benign retention the stomach can and would be cleansed. The grave suspicion should be confirmed by other evidences of cancer, and by the absence of the signs of a disease which might be accompanied by the conditions essential to lactic acid formation.

Lactic acid formed by bacilli in the stomach is always a pathological product, and its detection is consequently very important in diagnosis. Two tests are commonly used—viz., the iron test of Uffelmann and the oxidation aldehyd test of Boas. The reaction of Uffelmann, which is qualitative, usually suffices. When this reaction has been repeatedly positive it may be confirmed by the test of Boas, which is qualitative and quantitative. A quantity of lactic acid which does not give the Uffelmann reaction has very little diagnostic significance. It is a waste of time to make either test when free hydrochloric acid is present.

Uffelmann's Method.—Uffelmann recommended a clear, amethyst-blue solution containing carbolic acid and chlorid of iron. The reagent may be prepared by adding ten c.c. of a four per cent. aqueous solution of carbolic acid to 20 c.c. of distilled water containing one drop of the official solution of the chlorid of iron; or it may be prepared by dissolving four drops of pure liquefied carbolic acid in 20 c.c of distilled water and adding one drop of the solution of the perchlorid of iron. The reagent must always be prepared at the moment when the test is made. The blue only serves as a contrast color, and the reagent may be prepared by omitting the carbolic acid, the characteristic reaction being the yellowish-green color produced by the formation of the lactate of iron. It is recommended that the reagent be prepared by adding five drops of a ten per cent. dilution of liquor ferri chloridi (chemical reagent) to 50 c.c. of distilled water. This solution is clear and its color is imperceptible.

The test is made by using either the filtered contents or an ether extract of the same. When the filtrate is employed, the method of Kelling is to be preferred. Five c.c. of the filtrate are diluted to 50 c.c. by means of distilled water, and one or two drops of a five per cent. solution of sesquichlorid of iron (better, two drops of the ten per cent. dilution) are added. The yellowish-green tinge indicates the presence of lactic acid; or the reaction may be made by employing the solution containing two drops of the ten per cent. dilution of the official liquor ferri chloridi in 20 c.c. of distilled water. Five c.c. of the filtrate are added to 20 c.c. of the reagent, and the coloration is noted.

The liability to error is diminished by first extracting the lactic acid from the filtered contents with ether, and by testing the ether extract. Two methods may be employed. Five c.c. of the contents are shaken with five times their quantity of ether. After separation, the clear ether is decanted. Five c.c. of the ether extract are shaken with 20 c.c. of the iron reagent (containing two drops of the ten per cent. dilution). If lactic acid is present in the ether extract, the lactate of iron formed colors the water the characteristic straw-green. Or the method of Strauss may be used: A glass cylinder, graduated at 5 and 25 c.c. and fitted with a stop-cock, is used. Five c.c. of the filtered contents are first added, and thoroughly shaken with ether which has been added until the level of the fluid has been brought to the 25 c.c. mark. After standing until separation takes place, the stop-cock is opened, and the filtered contents and

the ether are let run out until the ether is lowered to the 5 c.c. mark. The cylinder is next filled with distilled water to the 25 c.c. mark. Two drops of a ten per cent. dilution of liquor ferri chloridi are added, and the coloration is noted.

The method of de Yong is simple and excellent. One or two drops of HCl are added to five c.c. of the filtered gastric contents, which is slowly evaporated to a syrupy consistency, permitted to cool, and extracted with ether. Five c.c. of distilled water are heated to the boiling-point, and the ether extract is slowly added. The ether is driven off by the heat of the water, and the extracted lactic acid is left in solution in the same quantity of distilled water as the quantity of gastric contents originally employed. One drop of a five per cent. solution of chlorid of iron is added after the solution has become cold, and the intensity of the greenish-yellow coloration is compared with that produced by adding one drop of the iron solution to five c.c. of solutions of lactic acid varying in strength from 0.5 to 4.0 : 1000.

All these methods, when properly followed, give trustworthy results. The yellowish-green coloration is slight when lactic acid is present in 1 : 3000; but it is very clear when 1 : 1000 lactic acid is present. The reaction is, consequently, sufficiently sensitive for practical purposes, for a smaller quantity of lactic acid than is revealed by the test possesses very little diagnostic significance. It is strongly recommended, for the purpose of a comparison, that the same relative quantity of contents (five c.c.) be always employed, and that the reagent contain the same quantity of the iron chlorid (two drops of a ten per cent. dilution of the liquor ferri chloridi to 20 c.c. of distilled water). The peculiar yellowish-green coloration only is characteristic of lactic acid. It is advisable to make a control test with five c.c. of a solution of lactic acid (1 : 1000), using the same reagent in both tests.

A number of substances give a similar coloration to that of lactic acid—cyanid of potash in the saliva (color remains after the addition of HCl but is discharged by corrosive sublimate), alcohol, sugar, phosphates, carbonates, oxalic acid, tartaric acid, butyric acid, etc. The coloration, when these substances are present in sufficient quantity, resembles, but is not the same as, that of lactic acid. It is always best to use the ether extract, and to make a control test. Under the proper precautions, the Uffelmann reaction is thoroughly trustworthy, and suffices, ordinarily, for all practical purposes.

The Method of Boas.—The principle of Boas' method, which is both qualitative and quantitative, is very simple. The lactic acid is converted by oxidation, under the necessary precautions, into acetaldehyd and formic acid. The acetaldehyd is tested for and estimated by using Lieben's reagent. The process is very long and tedious, and had better be submitted for its performance to an expert chemist. The qualitative analysis requires about two hours, and the quantitative about three hours, for its completion. It is an excellent method of research, which we find it rarely necessary to employ in practice.

(*b*) **Butyric Acid Fermentation.**—There are several bacteria which produce butyric acid out of carbohydrates: The bacillus butyricus (Prazmowski) is a very common form, and is very active in its movements, anaërobic, and builds central spores, around which the cell swells and lets the spore escape at the end, after enveloping it like a capsule. In starch and sugar solutions, and out of lactates or lactic acid, it forms butyric acid, hydrogen, and carbonic acid. With iodin, like starch ("amylobacter"), it stains—except in sugar solutions—deep blue. It does not liquefy gelatin. The bacillus butyricus (Hueppe) also builds central spores, but is aërobic, and liquefies gelatin and coagulates milk without changing its reaction. Out of lactates and lactic acid it builds butyric acid.

$$\underset{\text{(Lactic Acid)}}{2(C_3H_6O_3)} = \underset{\text{(Butyric Acid)}}{C_4H_8O_2} + 2(CO_2) + 4(H).$$

The oïdium lactici is abundantly present in some cases of retention with no free HCl. It forms butyric acid and hydrogen. The formation of hydrogen in the stomach disappears with the germ, and it also forms hydrogen in the fermentation tubes. Butyric acid may be split from fat by the secretion of the stomach (probably a ferment), or may be introduced into the stomach from without. The accumulation of the acid in the stomach is due to stagnation or to retention. The formation in the stomach is conditioned by stagnation and by a diet which furnishes the germs with a proper soil. It occurs often in association with lactic acid fermentation, the butyric acid being formed out of the lactic acid. Butyric acid is also formed in stagnation accompanied by normal or excessive HCl secretion. The butyric fermentation is not determined by the disease, but by the stagnation, by the supply of germs, and by a proper soil. It occurs in the mild form of stagnation and in retention, and regardless of the degree and the form of acidity. It is most common in

chronic gastritis, cancer, myasthenia, and pyloric obstruction. Butyric acid is a very strong irritant to the stomach, and is also toxic. Butyric is the most frequent form of acute gastric fermentation, but the formation of butyric acid is dependent upon the diet and upon little extraneous accidents rather than upon any particular disease of the stomach.

Butyric acid may be detected by its characteristic odor, which presents the most delicate test for it. Like the other volatile acids of the contents, it reddens a moistened blue litmus paper held in the end of a tube in which some of the contents are being boiled. It may be detected by distillation or by extraction with large quantities of ether, and by adding to the distillate or residue a large quantity of chlorid of calcium powder. Oil-drops are thus formed having the peculiar rancid odor.

(*c*) **Acetic Fermentation.**—Acetic fermentation is commonly due to the mycoderm aceti, which forms only surface colonies, and below a temperature of 35° C., and, consequently, can not take place in the stomach. But this form of fermentation does occur in the stomach, and is produced by other germs.

Acetic fermentation is quite frequent in alcoholism, especially when new, incompletely fermented drinks have been taken. The conversion of ethyl alcohol into acetic acid is an oxidation process, aldehyd being a middle product.

$$C_2H_5OH + O = CH_3COH + H_2O.$$
$$CH_3COH + O = CH_2COOH.$$

This form of fermentation may occur whenever there is stagnation or retention or when the stomach is kept supplied with alcohol. Acetic acid is also a by-product of yeast fermentation, and is often found in the stomach-contents which contain an excessive quantity of free HCl.

The acetic acid may be detected by its peculiar odor; or the distillate may be tested by exact neutralization with soda solution, the acetate formed producing a blood-red color in a dilute solution of chlorid of iron. This reaction is not sufficiently sensitive to be often positive in testing the gastric contents. In the absence of butyric acid, the reddening of the moistened litmus paper held in the end of a tube in which some of the contents are being heated may be attributed to acetic acid.

(*d*) **Alcoholic Fermentation.**—Alcoholic fermentation is due to the action of particular forms of yeast, which make alcohol ($2C_2H_6O$) and carbonic acid ($2CO_2$) out of glucose ($C_6H_{12}O_6$).

This form of fermentation, accompanied by the production of acetic acid, tartrates, etc., takes place in the stomach not altogether regardless of its acidity. It is almost arrested by one per cent. acetic acid, 2.5 per cent. lactic acid, 0.1 per cent. butyric acid, or 0.2 per cent. free HCl; and one-half of these percentages arrest the growth and begin to influence the action of the ferment. In the stomach the influence of the acidity is almost null.

Yeast fermentation may occur in any disease of the stomach, accompanied by motor insufficiency, be the acidity of the contents what it may. But the yeast is most active in retention. The alcohol yeasts consume oxygen, and, when this is not free in the fluid, they remove it from the sugar molecule. The fermentation is purest when air is excluded, but the yeast loses in vitality. The by-products are formed in greatest quantity when the yeast begins to degenerate and to die. The following equations represent this deoxidation process:

$$C_6H_{12}O_6 = C_6H_{12}O_2 + 4(O) = 2C_2H_6O + 2CO_2.$$

Dextrose, maltose, and levulose are directly fermentable. Cane- and milk-sugar are first converted into one of these substances by a ferment produced by the yeast. Starch must first be converted into maltose or dextrose by a diastatic ferment or by acids.

Alcohol may be detected by adding to the filtered contents a few drops of saturated solution of iodin in a solution of iodid of potash in distilled water, and by dropping in a solution of caustic potash until the brown coloration entirely disappears. The test-tube is then placed in a vessel containing hot water, and both are allowed to cool slowly. After a few hours a yellow precipitate of iodoform falls, which may be recognized by its odor and by the regular hexagonal crystals. Acetone gives the same reaction. Or the alcohol may be extracted from the filtrate by alcohol-free ether, and the test made with Lieben's reagent.

Putrefaction.—Putrefaction is a reduction process. It is due chiefly to anaërobic bacteria, and to such as extract their oxygen from the chemical compounds of the culture soil, from the fats and the albumins. The products of this germ reduction are not so simple as those of ordinary decomposition, which is an oxidation process. These reduction bacteria, which produce stinking products, are not uncommon in pronounced degrees of motor insufficiency.

Putrefaction may be accompanied by the formation of a

number of gases—NH_3, H_2S, N, PH_3, CH_4, etc.; a number of acids—butyric, acetic, formic, lactic, oxalic, etc.; a number of basic substances—leucin, tyrosin, skatol, ptomaïns, toxalbumins, etc.; and a number of ammoniacal compounds. The acids may be combined with organic bases.

The formation of H_2S, which seldom occurs in cancer, and only in some of the cases of retention, is the most important diagnostic product of gastric putrefaction. The H_2S is formed by reduction of the organic sulphur-containing compounds. It may also be formed by reduction of the sulphates, when it has no pathological significance. It is found in benign retention, regardless of the HCl and lactic acid percentages, after a diet rich in the organic sulphur compounds. A number of germs which find their way into the stomach may produce it, and among these germs is the bacillus coli communis (Lesage, Strauss). It may be detected by its odor or by means of alkaline acetate of lead. The lead papers are made by saturation in an alkaline solution of sugar of lead. The paper, previously wet in an alkaline solution, is hung free in a stoppered bottle containing the gastric contents; and, after a variable interval, it becomes brownish-black. If the action be too prolonged, the white sulphate of lead may be formed, and the reaction may be no longer apparent. Or the sugar-of-lead-cotton method of Schrank may be used.

Putrefaction without H_2S formation may be recognized by the stinking odor of the contents, and rarely occurs in the human stomach except in retention. Leucin, tyrosin, indol, and similar putrefaction products may be formed in the stomach when this organ contains pancreatic juice which has been regurgitated through the pylorus, or which has found its way into the stomach through a pancreo-gastric fistula. Nearly all the germs of putrefaction are gas-builders, and live preferably on the carbohydrates when the stomach contains them, although fermentation and putrefaction may coexist. Consequently, gastric putrefaction is conditioned by the existence of retention and by the presence of albuminous food and putrefactive germs. Putrefaction is increased by albumin digestive products, by fermentable matter, by small quantities of hydrochloric and lactic acids, and by pancreatic juice.

4. Gas-formation.—A number of gases are formed in the stomach by fermentation and putrefaction; but the flatulency thus produced can not be distinguished by the physical examination and the subjective symptoms from flatulency produced in other ways. Clinically, it is impracticable to measure the exact quantity of gas in the stomach, and to determine by

its chemical analysis whether we have to deal with swallowed air, with chemically generated gas, or with gas formed by germs. The most rapid and the easiest method is to make a test of the gas-forming power of the contents of the stomach, whenever and however obtained. For this purpose the vomit or the contents removed during digestion and during the period of repose of the stomach may be used; but in retention the contents removed before breakfast in the morning should be preferred.

The gas-formation is dependent upon the composition of the medium or soil. Peptones and other digestive products increase it, and it does not occur, at least to a notable extent, without carbohydrates. Consequently, on account of the possible exhaustion of the nutritive material in the contents, dextrose and peptones should be added to the tube cultures.

The cause of the gas-formation in the tubes is a living germ. The filtrate does not produce it. The sterilized precipitate added to the filtrate is also inactive. The unfiltered contents should always be employed in the tests. In the three-layer contents of retention, the gas production is most active in the bottom layer containing the numerous germs.

The activity of the gas-formation is dependent on the vitality and on the number of the germs which accumulate in the stomach. Gas-formation is moderate in stagnation, and often does not begin in the tubes until the end of one to four days. Flatulency is liable to be one of the symptoms in all the diseases of the stomach accompanied by stagnation; but in retention the gas-formation is active and rapid, and often the tube is nearly full of gas at the end of twenty-four hours. This is an important sign of retention, whether it be due to myasthenia or to obstruction.

Gas-formation is not prevented by the hydrochloric or lactic acidity of the contents; consequently, it occurs in both benign and malignant retention. It is impossible to say whether the excessive secretion favors gas-formation or whether the excessive secretion is a result of irritation. The germs seem to be virulent forms adapted to the acid medium. They progressively neutralize the cultures acidified with HCl, again become very active on the exhaustion of the HCl, and finally degenerate and become very sensitive to the anti-fermentative influence of the acid. In estimating the diagnostic value of the fermentation tests, the diet of the patient should be taken into account. A meat or proteid diet may almost

completely arrest it, unless putrefaction occurs; but the gas-forming fermentation recommences with the resumption of a mixed diet. A diet rich in carbohydrates increases the fermentation. The test should be made while the patient is on a mixed diet, is taking no germ-destroying drugs, and is employing no remedies to control the fermentation. The vitality of the germs and the degree of stagnation or of retention will thus be displayed most clearly.

The fermentation test may be made by using a Dunbar tube, a urine saccharometer, a Botkin bottle, or the stoppered test-tube of Moritz. The apparatus of Moritz is simple, easily cleaned, and readily filled. An ordinary test-tube is fitted with a perforated rubber cork. Through the perforation a glass tube, which is bent at two right angles or in a half circle, passes to the interior of the test-tube. The sterilized test-tube is filled with the well-mixed, unfiltered, and sweetened contents containing peptones or albumoses, the cork is pushed in, and the glass tubing is thereby filled with the displaced contents. The apparatus is inverted in a beaker and placed in the thermostat at 37° C. The gas, as it forms, collects in the test-tube. The mixture of gases may be analyzed chemically, but the quantity and the rapidity of its formation have a much greater diagnostic significance than has the composition of the mixture of gases. Some information as to the nature of the germ-growth may be obtained by examining a hanging drop prepared from the contents of the test-tube, and by comparing the result with the predominant forms of germs found in the contents soon after their removal from the stomach.

The other products formed by germs in the stomach have no diagnostic value. The study of the toxicity of the contents is of great interest in the pathology of the stomach, and the bacteriological examination is a fruitful field for original research. The ordinary methods of bacteriology may be readily modified so as to meet the special requirements.

CHAPTER V.

THE ANATOMICAL SIGNS.

The direct anatomical signs of the diseases of the stomach are very few. They may be found in the vomit, in the expressed contents after a test-meal, and in the water used to wash out the stomach. These anatomical signs are blood, epithelia, leukocytes, pieces of the mucous membrane, and small fragments of neoplasms.

The presence of blood in the contents or in the washings of the stomach may very readily lead to false conclusions. A small quantity signifies very little (unless it is present persistently or very frequently), and may be due to retching, to the introduction of the tube, to a temporary or a chronic congestion. Gastric hemorrhage occurs also in severe anemias and in cirrhosis of the liver. But the hemorrhagic diseases of the stomach are ulcer and carcinoma. Small quantities of blood are often found mixed with mucus in cases of gastritis, and this occurs so frequently as to be of some diagnostic value.

If the macroscopic and microscopic examinations of the contents do not make the presence of blood clear, it is necessary to use special tests. Spectroscopic examination is not clinically practicable. The two best tests for blood in the stomach-contents are those of Weber and Jaworski. It should not be forgotten that blood may be eaten with the food, or that iron (as a drug or as a compound of the food) may be swallowed.

Weber's test is a modification of Van Deen's. To ten c.c. of the filtered contents about three c.c. of glacial acetic acid are added, and the coloring matter of the blood is extracted by shaking with about five c.c. of ether. If blood is present, the ether extract is brownish; if the ether extract is uncolored, there is no blood. The separation of the ether may be facilitated by the addition of a few drops of alcohol. Add to the brownish decanted ether extract ten drops of fresh tincture of guaiac and about 20 drops of old spirits of turpentine or a small quantity of peroxid of hydrogen. After vigorously shaking the mixture for a while it becomes dark blue if blood is present. If there is no blood present, the mixture often becomes reddish-brown with a tinge of green. When the reaction is not clear, a little water should be added, and the coloring

matter extracted with chloroform. If blood is present, the chloroform extract is colored blue.

The iron test of Korcynski and Jaworski is made in the following manner: A small piece of the colored and suspected sediment is placed in a porcelain capsule, with a pinch of chlorate of potash and one or two drops of concentrated HCl, and is slowly and gently heated to drive off the chlorin. The procedure is repeated, after the addition of another drop of HCl, until the residue of evaporation is decolorized. The addition of one drop of a one per cent. solution of potassium ferrocyanid gives a Prussian-blue color if blood is present.

In the morning washings of the normal stomach are found a few single cylindrical cells, and, rarely, a few lymphocytes. If the fasting stomach contain free hydrochloric acid, which is a pathological sign, the spiral bodies of Jaworski and groups of the nuclei of leukocytes may often be found. In gastritis with excessive secretion these cell nuclei are numerous, and are mixed with the mucus, with the chief cells, and with mononuclear leukocytes. In asthenic and atrophic gastritis no spiral bodies are found, and no nuclei of cells the protoplasm of which has been digested, but numerous cylindrical cells and mononuclear leukocytes and sometimes beaker cells. Rarely, groups of cancer cells are detected. In suppurative gastritis and in perigastric abscess, large numbers of polynuclear leukocytes in the morning washings or in the vomit may reveal the nature of the trouble.

Exfoliated pieces of the mucous membrane are sometimes found in the morning washings, and this anatomical sign is most frequent in ulcerative gastritis (erosions). Pieces of the mucous membrane or of tumors may sometimes be found in the expressed contents, having been scraped off by the tube. This regrettable accident occurs very rarely when the velvet-eye tube is used, but the little fragments should always be searched for in the contents, and should be utilized for diagnostic purposes. The fragments rarely extend through the glandular layer, but sometimes do so, and, when hardened, cut, stained, mounted, and examined with the microscope, may give the anatomical diagnosis in a manner which leaves no room for doubt; but it should be remembered that a normal piece of the mucous membrane does not exclude anatomical disease of another part of the stomach, and that different forms of gastritis may coexist in the same stomach. The functional signs must make clear the predominant features of the inflammation, which may be asthenic or hypersthenic. Gastritis may also be a complication of ulcer or of cancer.

Bile and pancreatic juice are sometimes found in the contents, whether vomited or removed after a test-meal, and in the morning washings before breakfast. This may occur when the stomach is normal. Pathologically, the regurgitation of bile may recur persistently, and this would make obvious the easy passage of the pylorus and suggest the possibility of an obstruction of the duodenum below the opening of the common duct; but under such circumstances it should not be concluded that the pylorus is not the seat of an anatomical disease, for the pylorus may remain patent, like a rigid tube, in cancer, in cases of ulcer, and in cicatricial deformity.

The anatomical or dynamic or exact nature of a disease of the stomach may often be made plain by the results of treatment, even when all other signs fail to clear away the obscurity. This constitutes the therapeutic diagnostic test, which is applicable to both the primary and the secondary diseases of the stomach. The principles of the test are very simple. If the hypothetical diagnosis be correct, certain results should be obtained by a particular method of treatment, which would not give the same results in another disease; or the effects of treatment may be better or may be worse than would be obtained if the hypothetical diagnosis were true.

SECTION III.

GENERAL MEDICATION.

THE revelations of the diagnostic methods, together with our knowledge of the genesis and evolution of the diseases of the stomach, furnish the indications to be met by medication. Corresponding to the modern methods of diagnosis are the modern methods of treatment. The more accurate and complete knowledge of physiology, of pathology, and of the action of remedies has increased our ability to do good or to avoid doing harm. The pathology of the stomach, it is true, seems to have a greater charm for the lover of research than has the treatment of its diseases; but the interest of the patient begins with the medication intended to cure or to give relief.

The combination of remedies to meet the special indications of the particular diseases will be considered in Sections IV and V. The general medication consists in the use of remedies suggested by the condition of the patient and by the information given by the various diagnostic methods. The subject will be discussed under the following divisions:

1. Digestive hygiene.
2. Diet.
3. Physical remedies.
4. Symptomatic treatment.
5. Physiological treatment.
6. Bacteriological treatment.
7. Chemical treatment.

All curative treatment demands the removal or the control of the cause of the disease. This general principle of therapeutics has a very extensive application in the treatment of the diseases of the stomach; for a large number of these diseases are secondary, and the particular cause or causes of the primary diseases are often revealed by the clinical history. A man's stomach is no better and no worse, as a rule, than he himself makes it.

CHAPTER I.

DIGESTIVE HYGIENE.

DIGESTIVE hygiene should begin at birth and continue without a break throughout life. In this manner the primary diseases of the stomach could be prevented and life be made longer, more useful, and more comfortable. A strong stomach is not only a valuable possession, but it is a preventive against disease, and good digestion often decides the final result of a struggle against a dangerous disease. Digestive hygiene consists in giving to the stomach proper protection, proper repose, proper exercise, and proper work to do at regular periods. The history of the human stomach is, ordinarily, a long story of abuse, and pathologists rarely find a healthy stomach when the patient has died after the middle of life. However important digestive hygiene may be in the prevention of disease, it is absolutely essential to the cure of the diseases of the stomach.

In the general management of the diseases of digestion there is nothing more essential than a suitable environment—physical, moral, and social. These patients are very sensitive to cold, and become very languid and bilious in warm climates. Their bodies magnify every change of temperature. As a general rule, a moderately cold, dry location, of medium elevation, is very suitable, giving fresh air and sunshine and permitting outdoor life. The abdomen should at all times be well protected by the clothing, and left free to enjoy unimpeded the movements of the diaphragm.

The moral atmosphere is magnified in its gloom and decreased in its brightness by the delicate senses of these patients. The mind is particularly sensitive to the dark colors of life. There is nothing more depressing or injurious to such patients than the companionship of pessimists. The moral atmosphere should be sustaining, dissipating forebodings and inspiring contentment, hope, and courage.

The social atmosphere is another element of help or of injury. The slavery of the social life may offset all remedial influences. The performance of social duties under strain or exhausting excitement may leave no energy for digestion. It is often essential to place those suffering from a disease of the stomach where they can be free and can get rest and

adhere strictly to a proper diet. In these respects treatment in a sanitorium presents many advantages.

Rarely is the stomach injured by non-penetrating wounds of the abdomen; but diseases of the stomach, particularly ulcer, may be caused by traumatism—by a blow or by repeated or prolonged and strong compression. In this manner disease of the stomach is sometimes produced in shoemakers and in others who use the abdomen for holding objects or instruments in position; but more frequently the movements of the stomach are impeded or the stomach is displaced by prolonged compression and constriction of the waist. In this respect modern fashions are a curse to women. During the treatment of the diseases of the stomach, the action of these causes of disease must be excluded and the stomach be protected against external injury and compression. This hygienic rule is imperative during the digestive period.

"A person digests as much with his legs as with his stomach," wrote Chomel. Exercise facilitates nutrition, increases the bodily waste and needs, promotes the appetite, and, under proper conditions, is an aid to digestion. The healthy manual laborer awaits with impatience the hour of his meal, and eats all the more on account of his hard work; but if he becomes greatly fatigued, the appetite is lost. Sedentary habits may cause disease. On these points there is no difference of opinion.

When there is a serious disease of the stomach the matter is not so simple, and the prescribing of even moderate exercise during the period of digestion may be a grave error. Exercise creates a new demand for nutriment, and is beneficial on the condition that this demand be met easily and without injury. Patients with diseased stomachs are often unable to do this.

The action of exercise on the functions of the healthy stomach during digestion is positive. Moderate exercise hastens digestion, the stomach completes its evacuation a little earlier, digestive products do not accumulate in the stomach, the acidity of the contents is normal, and the movements of the intestines are more active. Violent exercise and hard labor are more decided in their action and the acidity of the contents is ordinarily below normal. In health it makes little difference whether a person take moderate exercise or rests, for neither is likely to derange digestion. Rest, in health, increases the acidity of the con-

tents, the digestive products accumulate in the stomach, and the motor function is less active than during moderate exercise. In sleep these variations are greater than in rest.

The influence of exercise, of rest, and of sleep is much greater in the diseases of the stomach than in health, and no rule can be formulated which is applicable to all these diseases. Individualization is the best plan.

The gastroneurasthenic should rest after his meals; for otherwise what is needed by the stomach may be used by other parts of the body and by the mind. The weak and the nervous should rest both before and after their meals, and the greater the weakness and the irritability, the more stringent should be the rule.

Myasthenic patients may or may not demand rest. If the myasthenia is not of a high grade (complete evacuation between meals) and if the organ has not been overloaded, the erythrism of the nervous system produced by exercise may be an aid to digestion; but observation proves that the highly myasthenic stomach commonly empties itself soonest under the gentler influence of repose. Exercise and the erect position increase the motor insufficiency, and may produce complete retention and an acute attack of pain, nausea, and vomiting. In all conditions, the digestion of a heavy meal should be begun with half an hour's rest; consequently, we almost invariably prescribe, in myasthenia, rest half an hour before and at least one hour after meals, and allow the exercise, in keeping with the individual indications, to be taken when the heavier work of digestion is over, and preferably in the open air. Some fondly imagine that they obey the laws of hygiene by promenading in their apartments or places of business. The exercise during the intervals of digestion should never produce greater fatigue than can be completely dissipated by twenty minutes of rest.

In many of the anatomical diseases rest during digestion is advisable. Gastroptosis, ulcer, hypersthenic gastritis, advanced carcinoma, and all diseases of the stomach accompanied by irritability of the mucous membrane or by emaciation and loss of strength, should be treated by repose, which should, in severe cases, be absolute.

The use of the voice immediately after meals is no less injurious than may be that of the muscles. The digestive tube is deranged by the unnatural respiration and by the compression between the diaphragm and the abdominal muscles. The abdominal tension is increased and the breathing performs a kind of massage. This rule is most often violated

by orators, professors, lawyers, ministers, and singers. There is no more pernicious habit than that of going directly from the table to the piano.

Cerebral is no less injurious than muscular fatigue: there are reasons to believe that it is more so. The overworked mind does not cry out in pain, but produces insomnia—it refuses to stop work. The strained muscle hurts, and muscular fatigue brings its natural cure—rest. The tired man is drowsy. The thinker should carefully select his food, let his brain rest while his stomach is hardest at work, and take regular and moderate exercise in the open air, so that the body will aid in demanding nutrition and rest for the brain. Nothing can equal the wisdom of these hygienic rules, except, perhaps, their perfect uselessness. Genius has long been condemned to live with a diseased stomach and will probably continue so to do.

CHAPTER II.

DIET.

"THE best, the only good, the only suitable, diet," wrote Trousseau, "is the one which the patient knows by experience best agrees with him." So notable has been the progress in the use of a diet in the cure and prevention of disease within the past twenty years that this proposition, though always false, is surely no longer tenable. The healthy man does not know how to feed himself, and the diseased man is far less likely to know.

A rational diet in the diseases of the stomach, through our more accurate and complete knowledge of digestion and nutrition, and of the various foods, is to-day possible. It is only within the past two decads that the great work of determining the functional power and the rich flora of the diseased stomach has been pushed on steadily to exact practical results. During the same period a flood of light has been thrown on the chemical pathology of nutrition, and on the utilization of food by the intestines in health and in disease.

But in addition to the increased knowledge of the functions and diseases of the stomach, and their relations to the work done by the intestines and to the state of nutrition, we are also in possession of more accurate knowledge of food. To-day

it no longer suffices to order a fluid, or light, or easily digestible, or nutritious diet; the physician must prescribe precisely and fully what the patient should eat. What is digestible and light and nutritious is unknown to most persons, who when left to follow blindly their diseased appetites and disordered sensations are very likely to eat too much, or too little, or the wrong things, or only a few articles, and to become morbidly introspective. The physician should select the diet in accordance with the indications furnished by the individual case, and should watch and control the effects.

The influence of the diseases of the stomach on nutrition is in practice a matter of very great importance. The clinic teaches, in a more trustworthy manner than experiments on animals, that the chemical work of the stomach, though very useful, is not necessary to the organism. The cases in the practice of every physician are very numerous in which the strength and weight of the patient are maintained, in spite of loss of nearly all digestive aid from the stomach. The utilization of the albuminous foods, starches, sweets, and fats may be complete in spite of the chemical inactivity of the stomach; but this is not because the stomach is useless, but because the organism is rich in resources.

There is no doubt that the intestines are capable of establishing complete digestive compensation when the stomach is insufficient, but on condition that a proper diet be given. The food will then be absorbed in an assimilable form, and will not be lost by fermentation or putrefaction; but this does not render it highly probable, as is claimed by some authorities, that the inanition in the diseases of the stomach (except in cancer) is due exclusively to deficient alimentation. Carefully selected diets may be well utilized, when the somewhat indiscriminate alimentation of health is not.

In a disease of the stomach without stagnation or excessive secretion there is little difficulty. The intestines, if the diet be correct, are fully equal to the possible extra work, when they are permitted to do it without hindrance; but where there is excessive secretion, duodenal digestion is interfered with and the action of the saliva is quickly curtailed. The fat which reaches the intestines is well digested and absorbed, but the carbohydrates and proteids are partly lost through fermentation and putrefaction, as the clinical diet guides indicate. In the diseases of the stomach with disturbance of the motor function, the stomach exerts its greatest power of doing harm. It may deliver to the intestines a chyme hardly fit for food, or it may reject the best kinds of food, or

withhold from the organism much of the food that is eaten; but if care be taken to secure delivery to the intestines of the right kind of food, in many of the cases nutrition may be well maintained, for the intestines may not have lost their compensating power. Disease of the stomach produces emaciation partly because the alimentation is improper.

Still, unquestionably in a large number of cases, the chronic inanition is due to deficient alimentation, the diet being too exclusive or too reduced in quantity through fear and ignorance.

In the dietetic treatment of the diseases of the stomach every effort should be made to keep the body well nourished; but food is not only nutriment, it is also a remedy. Not only must it meet the demands of nutrition and be appropriate to the digestive power of the stomach and intestines, but its physiological action must be such as to produce no harm, or to exert a remedial influence, or to give the diseased organ the appropriate exercise or rest. In the treatment of the diseases of the stomach, food is the most powerful and valuable physiological remedy. Consequently, a food should be selected, not on account of its nutritive value only, but also on account of its physiological action. Without a knowledge of the uses and action of food it is not possible to prescribe a rational diet.

In the first part of this chapter the rules for the selection of a diet in the diseases of the stomach will be deduced from the basic principles of alimentation. In the second part will be described the clinical evidences of a proper diet, or the clinical correction or confirmation of the diet as given by the reasoning of the physician. The first is a deduction from principles and from the subjective and objective signs revealed by the examination; the second is the testing of the conclusions by the daily observation of the individual case and the use of certain clinical methods of control.

I. SELECTION OF A DIET IN DISEASES OF THE STOMACH.

The grand aim of alimentation in disease is the restoration of the patient to a normal state of nutrition and to health. The object, then, is twofold—the nourishment of the patient and the exertion of a remedial influence on the disease. A diet which leaves either the one or the other out of consideration is fundamentally wrong.

The use of food as a remedy does not mean necessarily the

adoption of an exclusive diet, or so-called diet-cure. Dieting is sometimes made synonymous with slow starvation. A diet incapable of maintaining nutrition is a compromise enforced by very particular circumstances and endangers life when adhered to for a long time.

There is no general dietetic cure of the diseases of the stomach. Not only should the diet be suitable for the particular disease, but it should also be proper for the particular patient. Each case is a law unto itself, and demands as a basic principle of treatment individualization. No two patients are alike, either in the details of the disease or in their customs, habits, constitution, and state of nutrition. Neither is the dietetic treatment of a particular disease of the stomach fixed and invariable. In some cases it is advisable to change the accustomed diet, though it be improper, slowly and tentatively. Changes—and great changes—may be necessary, but they should be gradual and proportionate to the patient's power of adaptation. This matter demands special attention when the patient is old or weak. The same care is often necessary in making radical changes of the diet, as in going from a restricted diet to a more liberal one. The dietetic habits and digestive adaptations are sometimes too rudely disregarded.

Apart from the management of exceptional cases there are certain general indications to be met by the selection of a diet. These will be first briefly enumerated and then more fully discussed.

1. The quantity of the food should be sufficient to supply the needs of nutrition, and the composition of the diet should approach as nearly that of the normal diet as the variety of the disease of the stomach will permit.

2. Those foods should be selected which can be best digested and utilized, and which are least likely to ferment or decompose.

3. The physiological action must be such as to favor or to remedy the disordered functions and the anatomical lesions.

4. Not only the disease of the stomach but the state and power of other organs must be borne in mind.

5. The finances, habits, and peculiarities of the patient should be considered.

6. The directions should be complete and explicit, and should be changed to meet the daily indications.

1. *The quantity of the food should be sufficient to supply the needs of nutrition, and the composition of the diet should approach*

as nearly that of the normal diet as the variety of the disease of the stomach will permit.

Food converted into nutriment and absorbed by the blood-vessels, lacteals, and lymphatics is utilized in nutrition and in the production of force and of animal heat. But what is meant by nutrition? and how much food does the organism require?

The first theory formulated was that of oxidation or combustion. Soon after the great Lavoisier discovered oxygen and its properties were made known, and Regnault and other physiologists studied the formation of carbonic acid in the organism, it seemed almost sure that oxygen was carried by the hemoglobin of the red corpuscles to the tissues, and thus by combustion, more or less complete, produced urea, uric acid, carbondioxid, etc. This seemed to be a grand triumph for biological chemistry, and the great processes of nutrition and respiration were reduced to a simple chemical equation. Here is life revealing itself in its essence in the form of a chemical reaction. But further discoveries only produced confusion. The circulating albumin is the first to be destroyed, then the sweets, and lastly the fats. If nutrition be oxidation, the reverse should be true; and such easily oxidizable substances as alcohol and pyrogallic acid pass through the organism and appear unchanged in the urine. Moreover, the quantity of oxygen eliminated in combination with carbon, in the form of carbondioxid, is not in relation with the quantity of oxygen introduced. On the other hand, with an excess of food there is more oxygen absorbed and more urea eliminated. Also, when there is quite rapid loss of body fat, more oxygen enters. Again, oxygen alone is without action on albumins, sweets, and fats. The intervention of a third power is necessary.

The second theory is that of fermentation, and represents a transition stage. Nutritive exchange is not due to the introduction of oxygen into the system and to its action on the circulating nutriment, but is the result of the action of non-figured ferments in solution. Not only digestion but life itself is conditioned by the presence of ferments, since nutrition is cellular life, and cellular life makes up the life of the organism; but it is possible that the ferments widely distributed in the organism are the ferments absorbed from the digestive tube and the associated glands.

The third and probably true theory is that nutrition is the life and the function of the cell. Of this cellular theory there are two forms. Some contend that only the organized albu-

min undergoes transformation. In order that this view may be maintained in the light of recent discoveries, its supporters are forced to assume the partial regeneration of the albumin by the utilization of the analogous carbon and hydrogen elements of the sweets and fats. It is thus supposed that nutrition is somewhat like fatty degeneration. This theory is false, in that it contains only a part of the truth and is supported or supplemented by an untenable hypothesis. The dissociation, of which nutrition is the expression, affects both the protoplasm of the cells and the nutriment in circulation. Moreover, it is the circulating nutriment that is chiefly transformed, and for this reason the tissues are stable. Let this matter, on account of its great practical bearing, be examined more closely.

The fixed, organized "protoplasm" is changed only to a very slight degree in normal nutrition when the supply of nutriment is sufficient. When too few proteids, for instance, are introduced, the quantity of urea diminishes at once and if the condition continues the organism begins to live on itself; but this is unphysiological, and normally the quantity of organized matter varies but little. On the other hand, the quantity of circulating nutriment varies with the number of cells (for these are the active agents analogous to ferments), with the rapidity of the circulation, with the increase or decrease of the temperature, and also depends on the degree of special activity proper to each cell.

The life of the cell expresses itself in the ceaseless movements of its constituent elements. What are these elements? and what is the nature of these movements?

The arrangements, association, and constitution of the molecules of a cell endow it with its distinctive attributes and properties. It is in this moving molecule of protoplasm that the nutritive changes incident to life are expressed in their simplest form. It is in this "primordial basis," as Huxley has happily named it, that we shall hope to detect the intimate nature of nutrition. Simple and primitive in its life, the protoplasmic molecule is complex in its chemical composition and its constitution. First are the albuminous substances, with their four or five sorts of atoms, C, H, N, S, O, and sometimes also Fe or P; then come also the ternary, or C, H, O compounds, and lastly water and inorganic salts.

It has already been stated that the interior of this elementary molecule is in ceaseless motion; but motion presupposes force, and the regeneration of force means transformation, and transformation predicates a pre-existing substance.

Hence, the existence and continuity of cellular movements —or, in other words, cellular life—is dependent on the reception and transformation into living protoplasm, and on its utilization in the evolution of heat and force and in the removal of the by-products, of material fitted to undergo these changes. This received substance is called "nutriment," its appropriation by the living cell is known as "assimilation," and the expulsion of the waste is called "elimination." Assimilation, utilization in the formation of heat and force, and elimination constitute the nutritive changes, which are seen in their simplest form in the proper life of the cell. It will be noted that there is no suggestion of oxidation. The changes take place in the presence of oxygen as in the presence of water, but are the work of the cell itself.

Apart from its proper life the cell has a function to perform in nutrition. The one concerns itself alone; the other chiefly concerns the organism. This function is the transforming of the circulating nutriment into force and heat. This is, for the organism, the important work of the cell, producing by transformation of the circulating nutriment the force that is used in the performance of function. It is widely different from cellular nutrition, in that assimilation for the cell by the cell is absent. The one is continuous, persistent, innate; the other is intermittent, variable, produced by external excitation. Many cells, in addition to this general, have a special, work. The power to perform this special function is furnished by the organism; but the performance of each of these functions engenders force and animal heat. It is so with muscular contraction; it is also true of respiration, circulation, and digestion, and of the changes undergone by the nutriment in the liver and in the mesenteric glands before it reaches the general circulation.

Each cell, then, of the body may be considered as a center for the transformation, generation, and manifestation of force, being endowed with an impulsive movement which is its life. The new force is stored in its constituent elements, and is added to the unvarying radical and active force of the organism. It is in this way that the nutriment is utilized in nutrition, in the general meaning of the word, introducing into the body the potential food of life. But potentiality made active means transformation and disintegration. This is true regardless of the end of the generative power, be it used in cellular nutrition, in the interest of the organism, or in the performance of specialized work. Consequently, the nutritive process includes two grand results—the

formation of tissue and the evolution of force and heat; and the food, which is the source, has two corresponding destinies. The power of the organism to convert food into some form of force is limited. Only one class of foods—proteids—to even a limited degree increases dissimilation when introduced into the circulation in excess. When more food is assimilated than the organism requires, the excess is stored or organized. Be the excess proteids, sweets, or fats, the storage is practically almost exclusively in the form of body fat; but be any organ at the same time exercised or given, within limits, more work to do, that organ is strengthened and, if a muscle, also grows. On these observed sequences depends the possibility of improving and strengthening the organism by a fortifying diet.

In the dietetic treatment of the diseases of the stomach it is never an object to reduce the weight and strength. A starvation or an insufficient diet may be made temporarily necessary by very special circumstances, and it may be very difficult to decide, in the interest of the patient, how much is to be ultimately gained by injuring the organism in order to favor the stomach. There can be no question that the nutrition of the body must be improved, or as nearly maintained as the disease will permit. Consequently, except when forced to make a compromise, a primary necessity is the upholding or fortifying of the organism. To do this the quantity of food that the organism daily requires must be known. But this is not all. To select the quantity of food to be administered a knowledge of the nutritive value of food in the particular disease is also necessary.

The human organism as a unit may be considered a collection of centers for the transformation of force. This force comes from the outer world, and chiefly in the form of potential energy in the food. The difference between the force-value of the food as it enters the body and its remnants on their exit, represents the potential energy appropriated by the organism for the production of work and heat. It is not possible to measure directly the work done by the cell in the performance of its functions, which have to do with the preservation, perpetuation, and movements of the body, and with the mental and moral life; but by estimating the heat-value of the food as it enters and leaves the body we can obtain the quantity of force utilized by the organism, and expressed as so many units of heat; or by observing and analyzing the quantity of food necessary to maintain the equilibrium of the body, the daily needs of the organism may be expressed in

the alimentary principles. The results thus obtained serve as a basis for the quantity of food to be ordered, whether it be for the purpose of maintaining or of restoring the balance of waste and of repair or whether it be our object to reduce the body, to support life, or to force alimentation.

(*a*) **The Daily Needs of the Organism estimated in Units of Heat.**—To supply the daily needs of the organism and to maintain the balance of nutrition, enough potential energy in the form of food must be received to cover the consumption in the form of work and of animal heat. Such a quantity of food represents the diet of support. Under such a regimen as much is added to the body as is taken from it. The potential energy introduced and appropriated is exactly equal to the work and heat developed. The nutritive value of this merely supporting diet represents the daily needs of the organism, and affords for it an exact measure.

The amount of potential energy consumed by the body when at rest may be called the biological coefficient. The voluntary movements by which the body is placed in relation with external objects is not included in this conception. The store of potential energy is only drawn upon for internal work and for the supply of animal heat.

The quantity of potential energy necessary to maintain the dynamic equilibrium of the body in repose is dependent on the number of the noble elements of the body, and on their work in the maintenance of life. Each living cell is in continuous motion, and motion is an expression of transformed or liberated force. When these cells are more numerous, the consumption of potential energy is greater.

Not only the number of cells, but also the activity of the cellular protoplasm, which varies at different ages, must be considered. The adult requires more food than the child, and the needs of the body diminish with the degeneration of old age. The weight of the body is no measure of this need. The greater part of the body framework, the deposit of fat and other tissues endowed with little activity, neither consume nor liberate much force in their existence. A man, when emaciated, consumes less than when well nourished, because the number of noble elements—of cells which do a work for the organism—is diminished. The same individual on becoming fat, needs and consumes little more, because the fat cells have no active altruistic work to do.

The second factor of the biological coefficient is the potential energy consumed by the circulation, respiration, and the cells concerned in digestion and nutrition. One part is repre-

sented by muscular work and the other by glandular activity, and both are under the control of the most highly endowed dynamic cells of the body.

The smaller the mass, the greater is its relative surface area. Consequently, small animals lose more heat in proportion to their size than larger ones in a similar environment. The same law obtains with adults of different size, and applies also to the variations due to the growth of the infant into manhood. The consumption and the cellular activity are relatively greater in order to meet the proportionate by greater loss. Nothing can be more important than the protection of the body against the loss of heat in emaciated and in adynamic conditions.

The moisture of the climate is without influence, inasmuch as the diminished loss by evaporation is compensated by the increased loss by radiation and conduction. Heat production in the properly protected body in repose is constant. This is regulated through the nervous system by which heat production, established through the law of survival at a minimum, is transmitted as a fixed peculiarity embodied in a biological law.

The biological coefficient no longer represents the total work of the organism, as soon as the state of repose is broken. The amount of potential energy consumed is increased by the taking of food and by the process of digestion, by mental and muscular work.

The quantity of nutriment consumed is not proportionate, as was once thought, to the quantity of food introduced. The body is not a furnace that burns immediately and unceasingly everything combustible introduced into it. Digestion requires, through its muscular and glandular activity, an additional amount of activity, which varies with the digestibility and the physiological action of the constituents of the meal. Zuntz estimates that the amount of oxygen consumed is increased 15 per cent. during the digestion of a moderate meal, and a man digesting enough food to maintain the organism increases the consumption for twenty-four hours of rest and fasting by about ten per cent. The total production of animal heat is in like proportion increased.

This principle is of the highest practical importance. Mere excitants of the alimentary canal destroy potential energy. Food easily digested and quickly absorbed produces hardly a perceptible increase of nutrition waste. Only by a correctly selected diet can the reduced organism be restored to a good nutritive state. The introduction of food increases the waste

by its action on the alimentary canal, and not directly by being itself consumed. A rich diet may mean a loss, as there is no luxurious consumption. The excess is undigested, unabsorbed, or incompletely oxidized. Alimentation is most economical when the nutriment introduced is the exact amount required. An excess is stored or wasted, and when too little food is taken the body lives on itself.

Muscular work is the cause of the greatest expenditure of force above that of the biological coefficient. The increased consumption of oxygen in very slight movements and its demand in strained work illustrate how close and great is the influence of muscular work on the consumption of potential energy. Zuntz estimates the daily consumption by hard work at 25 per cent. above the expenditure in repose.

In the performance of muscular work, much more potential energy is consumed than is expressed by the useful work performed. Rubner's researches give a loss of nearly four-fifths. This loss is not a constant one, and is not so great when the man is fresh and trained to use his muscles. Fatigue, a relatively great effort, and a lack of training involve great loss and excessive consumption.

The following table gives in round numbers the daily consumption by an adult weighing 65 kilograms, the figures representing 1000 heat units :

Rest in bed, 1800 Cal., or 28 Cal. per kilo.
In repose, 2100 Cal., or 32 Cal. per kilo.
In light work, 2300 Cal., or 33 Cal. per kilo.
In moderate work, 2600 Cal., or 40 Cal. per kilo.
In strained work, 3100 Cal., or 48 Cal. per kilo.

The estimation of the needs of the organism in units of heat does not correspond with the actual uses of food in nutrition. Only a part of the food assimilated is used in the production of heat. Again, although the end-products of the fats and carbohydrates are the same in the calorimeter as in the body, those of albumin are different. The calorimetric value of animal albumin is 5.7 Cal., that of vegetable albumin is 5.6 Cal.; but the nutritive value of albumin is represented by the number of heat units liberated by its oxidation and conversion into the compounds which represent the forms in which it is eliminated from the body, and, according to Rubner, is 4.2 Cal. and 4 Cal., respectively, for animal and vegetable albumin. The nutritive value of ten gm. of fat is equal to 23 gm. of albumin or carbohydrates; or—

1 gm. albumin or carbohydrates = 4.1 Cal.
1 gm. fat = 9.3 Cal.

The conversion of the older estimate of the needs of the organism, in so many grams of albumin, fat, and carbohydrates, into the newer calorimetric equivalent is an easy process of multiplication; but the new method is clinically a great advance on the old one. The alimentary principles can, within limits, be substituted for one another, and in the treatment of the diseases of the stomach the substitution is usually and necessarily made. The calorimetric method renders a complex problem simple and easy. The force-value of the diet is revealed at a glance, and it is quickly seen whether it is insufficient, supporting, or strengthening.

(*b*) **The Daily Needs of the Organism as represented by the Alimentary Principles.**—The amount of force daily needed by the organism, both in a state of repose and in internal and external activity, can thus be accurately estimated in units of heat. The measure of the oxygen consumed, the difference between the heat-value of the food and that of the waste products eliminated, minus the amount retained and organized or stored in the body, and the estimation of the heat-value of the food necessary to preserve the body for a long period in nutritive equilibrium, have revealed with sufficient precision the needs of the organism. Turn we now to a short study of the available material for the supply of these demands. The daily needs of the organism must be supplied by that which is of nutritive value in the food.

The value of the food to the organism is dependent on its chemical composition, on the combination of the alimentary principles, and on its digestibility and utilization.

Not all the chemical constituents of a food are of nutritive value. A part may be insoluble in the digestive fluids, and may either become the food of micro-organisms or pass into the feces. A part may be absorbed, only to be eliminated on account of its worthlessness in nutrition. A part may waste as much potential energy in its digestion and assimilation as it supplies in its products. A part may be absorbed unchanged, and may be useful or essential to nutrition without directly contributing potential energy to the organism. These are only important on account of their chemical and physiological action. A part may be absorbed after preliminary transformation and be utilized in supplying the needed force and heat.

Food, then, contains (*a*) material which maintains the nutritive equilibrium and furnishes heat and force. The most important of the members of this class are the proteids, fats,

and carbohydrates. Alcohol and the organic acids are also capable of furnishing some force and animal heat in their consumption. Fat and albuminous foods may be either of vegetable or of animal origin, and possess nutritive values according to their qualities and chemical constitution. The carbohydrates are products of the vegetable kingdom. All these foods are organic.

The second class contains (*b*) inorganic compounds, or mineral matter. The chief of these inorganic elements are H, O, Na, K, Cl, Ca, Mg, Fe, Mn, P, and S. These elements are in various combinations as salts, and are as essential to the organism as are the members of the first class. There exists for each a minimum quantity, without which inanition, and even death, may result. These inorganic compounds are present in excess in all the foods employed in diet prescriptions. This is not the case with water or common salt; these will receive further consideration.

These compounds exist in all three kingdoms, but give no force or heat to the human body. Their function is almost exclusively a chemical one.

The remaining compounds (*c*) are organic, and owe their value to their physiological action. This value may be due to their odor or taste, and to their local action on the stomach or intestines, or on nutrition, or on one or more organs of the body. Some of them also furnish a very small quantity of energy, which may be consumed in the work of the body, as the alkaloids and glucosids. This class exists in all food that is not insipid. They give the pleasant odor and taste to food. Their physiological action is essential to good digestion and to nutrition.

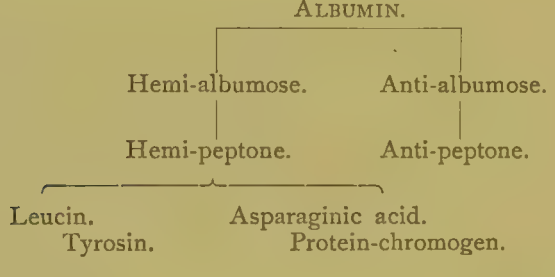
A fourth part of the food (*d*), important for its local physiological action, is the indigestible residuum.

The Uses of the Albumins.—Albumin may be absorbed without having been transformed by the digestive ferments. Albumins are, however, rarely eaten raw or in a fluid state, except in milk, and this fluid rapidly becomes a solid in the normal stomach under the coagulating power of the labferment. In health very little albumin is absorbed without transformation, although this may occur when a very large quantity is eaten. A part of it then appears in the urine, as also occurs when the albumin is introduced directly into a vein.

The only chemical work performed by the formed elements of the gastric juice is the digestion of the albumins. This transformation is a progressive one. The albumin is converted into syntonin, albumoses, and finally peptones—which

differ from one another in their degrees of solubility and diffusibility through an animal membrane and in their chemical properties. The transformation is in all probability a change of the chemical constitution of the molecule, while the chemical composition is not altered. Recent investigations would indicate that the albumin molecule is split up into smaller ones with the same chemical composition. Water here, as in all chemical processes, plays an essential part. Very little albumin is converted by the gastric juice into true peptone, the process not going further than the albumoses. With the pancreatic juice, however, this is otherwise. The intermediate compounds are rapidly changed into the end-products of albumin digestion, and so powerful is this ferment that a part of the peptones may be decomposed, and the greater part of the potential energy of the molecule may be wasted.

According to the generally accepted ideas of Kühne and his followers, the albumin molecule is split into two series of compounds of different qualities, to which the names anti- and hemi-albumose and anti- and hemi-peptone have been given. The following diagram represents this view:



These distinct and double but contemporaneous methods of division were first demonstrated by Kühne in the digestion of fibrin, and the same principle has been shown to obtain for other forms of albumin, such as gluten, legumen, vitellin, casein, myosin, etc.

The products of gastric and pancreatic digestion are not the same, even with the same form of albumin. In the stomach, albumoses are formed in far larger quantities. In the intestines the peptones predominate.

The nutritive value of these products of albumin digestion is variable. Consequently, the form of albumin prescribed, the location of its transformation, and the rapidity of its absorption are of practical importance. This is most evident in disease. In health, selected and digestible nitrogenous

food is nearly all absorbed, only three to five per cent. being lost in the feces. But the percentage of utilization varies widely with different articles of food in the disorders and diseases of the digestive tube.

The greater part of the products of the digestion of albumin are absorbed before the ileocecal valve is reached, only about one-fifth reaching the large bowel. This is the work of the cell, which pours these products into the blood-vessels exclusively. The lymph-vessels receive none.

The products of the digestion of albumin are taken up, on their passage through the wall of the alimentary canal, by the white blood corpuscles. None of these products are received free into the serum of the blood. This digestive leukocythemia is only present during the digestion of albuminous foods, when the number of white corpuscles in the veins is enormously increased. These products are absorbed by the epithelial lining of the stomach and intestines, and are taken up out of the alimentary wall by the white corpuscles and reconstituted into albumin. The white corpuscles assimilate albumin.

All the products of the digestion of albumin down to the true peptones are received by the white corpuscles and reconstituted into cellular protoplasm. The albumin thus becomes endowed with life and loses the personal identity it possessed before digestion and absorption. Out of this reconstituted albumin all the protoplasmic cells of the body receive the food which is to supply the wear and tear of life. The albumin of food is also used in other ways, and reappears in the albuminous constituents of the secretions. The casein of milk is a product of cell activity, as is likewise hemoglobin. The last is a synthetic compound of hemochromogen and of globulin. The mucin is a compound of albumin and of carbohydrates under the influence of the epithelial cells. In like manner many of the other cells build special forms of albumin out of the albumin of the food. The albumins constitute about one-eleventh of the weight of the body, and nearly one-half of the total quantity is contained in the muscular system.

The dissimilation of albumin is a complex process, which is best and almost exclusively known in its end-products. This constitutes the chief use of albumin, the disintegration liberating force and heat and demanding replacement by nitrogenous food. It is only when the assimilation of albumin is greater than its dissimilation that the excess becomes organized.

The quantity of albumin daily disintegrated by a well-nourished person weighing 70 kilograms is about 60 gm. when fasting. The quantity of albumin in a well-compounded diet should never be less than this amount. One gm. of fat is equal to about 2.4 gm. of albumin or carbohydrates, and the albumin can, within a wide range, be replaced by and replace the other two large classes of food; but the caloric value of the albumin should never be greater than one-third nor less than one-sixth of the caloric value of the other two combined. The albumin required by the body for organization can be replaced by no other kind of food.

On an exclusive diet of lean meat, about two and one-half or three times as much albumin as is disintegrated and eliminated from the body in hunger (50 to 60 gm.) is required to establish nitrogenous equilibrium. The average quantity is about 1200 gm. of lean beef. But while the nitrogenous equilibrium may be maintained on an exclusively meat diet, no increase of weight and strength can be produced, for the dissimilation becomes greater as the quantity of albumin is increased, and the equilibrium is re-established by more active metabolism. The increased ingestion of albumin renders the fluid which surrounds the cells richer in albumin, but no albumin is organized and the development of new cells is not stimulated. On an exclusive nitrogenous diet man neither fattens nor organizes albumin after the balance of nutrition is established. Consequently, a healthy body may be maintained, but a weak or emaciated person can not be made stronger. The first effect of an exclusive meat diet is loss of weight and strength. The body fat is utilized and burned, and the increased albuminous waste is greater than the quantity of albumin digested and assimilated. To establish good nutrition so large a quantity of meat is required that even the most tender and palatable meats become disgusting. The normal excitation of intestinal peristalsis is removed and the acidity of the contents of the small intestine disappears. Constipation, putrefaction, uric acid precipitation, and auto-intoxication become the prominent symptoms, and to prevent injury the body must be placed in repose, non-nitrogenous food must be administered with the meats, and eliminative treatment must be employed. Gradually, under rest, laxatives, and large quantities of water, the symptoms are relieved and the repugnance overcome. Eventually, nitrogenous equilibrium may be established as more and more meat is eaten; and the individual then becomes stronger, but no fat or albumin is organized.

Carbohydrates.—The carbohydrates, found only in traces in the animal body, exist chiefly as the sugars, organic acids, and starches in vegetable food and fruit. The starches must first undergo digestive transformation before absorption and utilization. Some of the sweets are already absorbable in the form in which they are taken into the alimentary canal.

Starch, when finely divided and liberated from its inclosing capsule of cellulose, is rapidly transformed by the ptyalin of the saliva, when the medium is alkaline, neutral, or slightly acid, into soluble starch, erythrodextrin, achroödextrin, and maltose. The starch which escapes salivary digestion, and the ptyalin products unabsorbed by the stomach, are delivered to the intestines for the completion of the digestive transformation by the diastase of the pancreatic juice. Theoretically, the end-product of starch digestion is maltose, but in the digestive tube the transformation rarely goes beyond the dextrin stage, maltose being found rarely and only in traces in the portal vein.

Milk-sugar probably requires and undergoes no digestion. Cane-sugar is inverted by the HCl of the gastric juice, and also by the intestinal secretion. Levulose, a digestive product of cane-sugar, and also found in fruits and honey, is absorbable. The sugars may nearly all be absorbed as such, and their digestive transformation increases their absorbability and liability to ferment.

The sugars introduced with the food and formed by the digestion of starch are absorbed by the radicals of the portal vein, and undergo further changes fitting them for utilization. If the analysis of the stools alone be taken in evidence, the carbohydrates are almost completely digested and absorbed, when properly prepared and not taken in excessive quantity. But practically and clinically, both in health and in disease, a considerable quantity undergoes fermentation. The products of fermentation may possess some nutritive value, and the slight acidity of the intestinal contents produced by the fermentation is a safeguard against albuminous decomposition. The gases of fermentation interfere, when in excess, with the functions of the bowel, and the organic acids may produce irritation and inflammation.

The absorbed products of carbohydrate digestion are assimilated, and are either stored as glycogen in the liver, muscles, and white blood corpuscles, or are burned as grape-sugar in the production of force and heat. There is also no doubt that the organism can and does, in certain conditions, transform the carbohydrate products into fat to be stored in the

body. The carbohydrates diminish nitrogenous waste, and, in very large quantities, reduce the daily nitrogenous loss below that of fasting. Fat is also protected by the carbohydrates, 240 gm. being isodynamic for this purpose with 100 gm. of fat taken as food. The amount of fat can be completely protected if with 100 gm. of albumin about 600 gm. of carbohydrates are digested and utilized. If the quantity of carbohydrates be increased, the body may become fatter and some of the albumin may be organized, but in order to insure the latter the quantity of albumin should also be increased. If fed exclusively on carbohydrates man would starve, through the uncovered loss of albumin, unless carried off earlier by the diarrhea and enteritis produced by excessive fermentation.

Fats.—The digestion and absorption of fat begin in the intestines. In the stomach only small quantities of fatty acids are normally found, and are either introduced as such or are separated from the triglycerid molecule by bacteria—and possibly by a special ferment. A special ferment of the pancreatic juice splits a part of the fat into fatty acids (93 per cent.) and glycerin (7 per cent.), the fatty acids combining with alkalies (of the bile, chiefly) to form soaps, and with the aid of these products, and of the bile and the pancreatic juice, the remaining fat is emulsified. Only about one-eighth of the fat is decomposed, but the relative proportion of the fatty acids and soaps increases as the lower end of the bowel is approached. This is due partly to the action of the splitting bacteria, but chiefly to the rapid absorption of the emulsion of neutral fat by the epithelium covering the intestinal follicles, and possibly by wandering leukocytes. The absorbed fat reappears in the lymph as neutral fat, and is utilized in the production of heat, of force, and of adipose tissue, which latter constitutes from 5 to 25 per cent. of the weight of the body. No other tissue is subject to such wide quantitative variations.

Fat alone is unable to check the waste of nitrogenous tissues, and only prolongs the period of starvation. A fasting man wastes more than twice as much fat as albumin, but protection against loss of fat can be given either by albumin, by fat, or by carbohydrates; both a loss of fat can be prevented and a gain of fat insured most economically by a judicious combination of the three alimentary principles. As a protective against loss of fat, 100 gm. of fat are equivalent to about 225 gm. of albumin. Large quantities of

fat can prevent loss of body fat, or even increase its quantity in spite of simultaneous albuminous emaciation.

The more fat is given, other things being equal, the more is found in the stools. But the total quantity of fat absorbed increases until the quantity administered is about 300 gm. This seems to be the limit of digestive power.

Gelatin.—Gelatin is a very valuable force- and heat-producing food. It is never organized into fat or flesh, and in very large quantity can not completely cover albuminous waste; but with gelatin the quantity of albumin needed can be reduced to a minimum, even below the amount of albuminous waste during fasting. As a protector of body albumin, 100 gm. of gelatin is equivalent to about 35 gm. of food albumin, or 200 gm. of carbohydrates. On the other hand, gelatin does not protect against loss of fat as well as do either fat or the carbohydrates, 100 gm. being equivalent to about 25 gm. of the dissimilated fat of the body.

Combinations of the Alimentary Principles.—When fats are combined with albumins, a little less than half as much albumin is required to maintain nitrogenous equilibrium as in an exclusively albuminous diet. If, after the combination is found which in an individual case exactly maintains the nutritive equilibrium, more albumin is given with a proportionately large quantity of fat, both albumin and fat will be organized and deposited. If carbohydrates be substituted for the fats, increase of albumin causes a slight increase in both the albumin and the fat of the body; increase of both results in bodily gain of flesh and fat. In protection against albuminous waste the carbohydrates are more powerful than the fats, but 100 gm. of fat are equal to 240 gm. of carbohydrates in the protection against loss of fat.

The body can be most economically nourished by a judicious combination of the three alimentary principles. The proportion of the caloric value of the albuminous to the non-albuminous foods, and of the fats to the carbohydrates, should be 1 to 3 or 5.

The following average estimates of the quantities of the combined alimentary principles required to maintain nutritive equilibrium are taken from the works of Uffelmann, Voit, Camerer, and others:

Age.	Weight.	Albumin.	Fat.	Carbohydrates.	Mother's Milk.
1 week,	Per kilo.	3.7	4.3	4.4	.
1 "	Total	2.5	10.2	17.5	250 gm.
2 weeks,	Per kilo.	4.4	7.4	10.	. . .
3 "	" "	4.8	5.6	5.7	. . .
4 "	Total	5.6	22.4	38.6	550 gm.
5 "	"	7.2	28.5	49.2	700 gm.
2 months,	Per kilo.	4.5	5.2	5.4	. . .
3 "	Total	9.2	36.6	63.2	900 gm.
4 "	Per kilo.	4.2	4.6	5.	. . .
6 "	Total	10.3	40.7	70.3	1000 gm.
1 year,	Per kilo.	4.	4.	8.5	. . .
2 years,	" "	4.1	4.	8.	. . .
3–6 "	" "	3.6	3.	10.	. . .
7 "	Total	55.	25.	180.	. . .
7–15 "	Per kilo.	28.	2.	10.	. . .
8 "	Total	60.	44.	150.	. . .
9 "	"	61.	47.	200.	. . .
10–15 "	"	75.	30.	250.	. . .
Adult (man, repose),	Per kilo.	1.5	0.8	7.5	. . .
" " "	Total	100.	56.	400.	. . .
" (man, light work),	"	110.	56.	500.	. . .
" (woman, " "),	"	90.	40.	400.	. . .
" (man, hard work),	"	110.	80.	500.	. . .
" (man, forced work),	"	145.	100.	500.	. . .

The following table gives the quantity of the alimentary principles in the foods most frequently ordered in the treatment of diseases of the stomach:

Food.	Albumin.	Fat.
Lean beef,	20.7	1.7
Beef-pulp free from fat,	20.	0.5
Medium beef,	21.	5.4
Fat beef,	16.7	29.3
Fat hind quarter,	20.8	23.3
Fat (steer) tenderloin,	18.8	16.7
Fat (heifer) tenderloin,	19.2	5.8
Boiled beef (medium),	34.1	7.5
Roast beef (medium),	34.2	8.2
Fat veal,	18.9	7.4
Lean veal,	19.9	0.8
Veal chop,	20.2	6.4
Broiled veal chop,	29.	12.
Fat lamb (quarter),	9.7	35.8
Lean mutton (quarter),	11.1	25.4
Mutton chop,	14.3	8.6
Breast of young chicken,	19.4	2.8
Lean chicken,	23.3	3.1
Pheasant,	25.3	1.4
Pigeon,	22.1	1.
Ham,	24.7	36.5
Scraped raw ham (lean),	23.5	1.5

Food.	Albumin.	Fat.	Carbohydrates.
Bouillon,	0.4	0.6	. . .
Expressed beef juice,	7.	0.5	. . .
Veal extract (bottle),	2.8	. . .	. . .
Beef " "	1.8	. . .	. . .
Oysters,	10.	1.5	. . .
Barley meal,	12.5	1.	75.
Flour,	13.6	1.	73.
Oatmeal,	11.6	5.	69.
Rice,	7.5	0.6	78.
Bread,	6.8	1.5	43.7
Dry toast,	9.5	1.	75.
Crackers,	7.5	5.	58.
Potatoes,	1.5	2.	20.
Mashed potatoes (milk),	3.1	0.9	21.3
Cauliflower,	2.5	0.4	4.
Asparagus,	1.5	0.3	2.5
Ripe grapes,	0.6	. . .	14.
Prunes,	2.4	0.5	60.
Sole,	11.9	0.3	. . .
Brook trout,	19.2	2.1	. . .
Carp,	21.9	1.	. . .
Mackerel,	19.4	8.	. . .
Pike,	18.4	0.5	. . .
Cod,	16.2	0.3	. . .
Spinach,	2.5	0.6	4.5
Apples,	0.4	0.8	3.
Grapes,	0.6	0.8	16.3
Strawberries,	1.	0.9	6.7
Honey,	1.2	. . .	73.7
Cane-sugar,	0.3	. . .	96.2
Beet-sugar,	2.3	. . .	93.9
Macaroni,	9.	0.3	76.8
Wheaten grits,	10.4	0.4	75.5
Graham bread,	9.	1.	50.

	Albumin.	Carbohydrates.	Alcohol.
Beer,	0.5	5.2	3.5
Bordeaux,		0.3	10–15
Rhine wine,		0.5	10–15
Tokay,		15-22	16–18
Champagne,		8.5–11.5	12.
Cognac,		. . .	60.
Brandy,		. . .	45.

A knowledge of the quantity of food required to meet the demands of nutrition is of value in many respects. It is the basis of a diet selected with a view to support, or to reduce, or to increase the weight and strength. It reveals by comparison the cause of emaciation in many cases of disease of the stomach—viz., an insufficient diet. It makes it possible to avoid excessive eating and overtaxing the digestive organs, and the oft-resulting myasthenia, stagnation, and fermentation.

There is no doubt that a strong, well-nourished body possesses a greater resistance and healing power than one which is weak and starving; but in the treatment of the diseases of the stomach an insufficient diet must often be prescribed. Short abstinence and the consequent complete repose of the stomach is an essential indication in the treatment of acute gastritis. Ulcer may also compel the use of an insufficient diet. Even the dynamic affections may render a more or less complete temporary withholding of food imperative or advisable. Myasthenia may demand a reduction in the quantity of food below what is necessary to support; for, as a general rule, the larger the quantity the longer is the sojourn, and it is always imperatively necessary, whenever possible, that the stomach should completely empty itself between meals.

The result of an insufficient diet is subnutrition, which renders it necessary for the body to live in part on itself. More than twice as much body albumin is dissimilated in proportion to the body fat when the patient is lean (1 : 4) than when the patient is fat (1 : 9). The total quantity of the blood is decreased, and the serum becomes poorer in albumin. The cells of the body do not lose their power of multiplying. This innate and vital function of the cells is only in abeyance, and again becomes active in proportion to the increased ingestion of food. This principle is well illustrated by the renewal of the body during convalescence from severe acute diseases. Subnutrition produces no injury if the latent recuperative powers of the patient are not lessened, and the diseased stomach may be favored without fear of permanent injury by subnutrition provided digestion and assimilation are so improved thereby as to render it easy to restore, at any moment, the loss of weight and strength.

It is often difficult to make the choice between reducing the body and injuring the stomach, for a blow aimed at one may strike both. The difficulty may often be overcome by reducing the nutritive demands to a minimum by absolute rest in bed, and by supplementary rectal feeding. But stomach rest usually means the selection and diminution of the quantity of the food so as to support the body in repose and to demand the least possible functional activity of the stomach. In some cases, on account of pain, or loss of appetite, or vomiting, it is difficult to obtain the ingestion or retention of enough food to maintain nutrition. In others, moral suasion and tonics will fail to secure the eating of enough food, or of the right kind, to build up the weight and strength.

The only valuable resource then left is artificial and forced feeding.

This is a very valuable method of alimentation in some of the diseases of the stomach. In anorexia nervosa forced alimentation may be employed with the prospect of obtaining excellent results. Whenever, in organic disease, the patient persistently eats too little food, the additional quantity should be introduced through the tube, if there be no danger in its employment and if there exist no reason why the additional food can not be digested and absorbed, as would be the case in myasthenic and in obstructive retention. Pain is another cause of inanition in the diseases of the stomach, the patient by gradual exclusion adopting a starvation diet. This is commonly the case in hypersthenic gastritis and the diseases which it complicates. In neurasthenia gastrica the digestive discomfort may produce a dread of food. Artificial feeding will, under these circumstances, be found a valuable remedy. The use of tube feeding may also be advisable in ulcer, where, on account of pain, the fear of food is great, or where vomiting becomes uncontrollable. If the stomach-tube be introduced to the lower third of the esophagus, or even just through the cardia, the food may be introduced through it without danger in ulcer, and with a good prospect of its retention.

It is a curious fact that food introduced through the tube is often retained when, if swallowed, it is invariably rejected by the stomach. In nervous vomiting artificial feeding is a valuable remedy, and may be the only means of obtaining the digestion and absorption of enough food to maintain nutrition.

Excellent preparations for the administration of food by the tube are combinations of milk, expressed meat juice, meat powder, raw eggs, sugar of milk, and bouillon. The following combinations are often well borne:

Milk, ... 500.
Meat powder, ... 30.
Milk-sugar, ... 20.

Or—

Bouillon, ... 300.
Meat juice, ... 50.

Or—

Meat powder, ... 30.
Two raw eggs.

2. *Those foods should be selected which can be best digested and utilized and are least likely to ferment or decompose.*

Each individual has his own opinion of the digestibility of the various foods. Physicians themselves are as much at variance about the matter as are the patients. Such a state is regrettable. False impressions and narrow theories, as they always do, have led either to bigotry or to agnosticism. The one rejects all investigations as useless; the other bends all facts to one theory. Both methods are equally disastrous to the patient.

The point of view has been wrong. The digestibility of food must be defined and studied from the point of view, not of hygiene or physiology, but of the physician. The value of food as a remedy is dependent upon its physiological action and upon the ease and certainty with which it undergoes transformation and absorption. In order to use food rationally as a remedy it must be known in a particular manner.

The duration of the sojourn of a food in the stomach is no full measure of its digestibility. The investigations of Gorse, Spallanzani, Beaumont, Richet, Busch, and Kühne only show the work of the stomach as a motor organ in certain pathological conditions. Gorse and Spallanzani (1785) made their observations on a ruminating patient. Merycism is a neuromuscular affection of the stomach, and the rapidity with which a food is passed through the pylorus gives no indication of the ease with which it undergoes digestion in health.

Beaumont (1838) made his observations on Alexis St. Martin, through a gastric fistula resulting from a gunshot wound. Gastrostomy had been performed by Verneuil on Richet's patient for a benign stricture of the esophagus. The adhesions of the stomach always disorder its movements, and the duration of the sojourn of a food in a pathological organ is no index of the influence of a given food, even on the motor function, in health. The investigations of Busch and Kühne were made through a duodenal fistula. In this case the motor function of the adjacent stomach could not be uninfluenced. Likewise, the experiments upon gastrostomized dogs show the action of food on the pathological stomach of a carnivorous animal, and are not applicable to the normal or the diseased human stomach. All these investigations throw some light on the action of certain articles of food on the movements of the stomach in certain pathological conditions. The tables of digestibility constructed out of such data are almost valueless to the physician and give, except when the guess is a happy one, altogether false results. The end of alimentation in health or in disease can never be attained so long as the influence of a food on the movements

of an unhealthy stomach is taken as a full measure of digestibility.

Still more valueless are the results obtained by artificial digestion, which give a false idea even of the resistance of the food to digestive transformation. The stomach is not a dry, motionless receptacle with impermeable walls.

The way to scientific results was opened by Leube. The digestibility of food in disease of the stomach is displayed by the manner in which the food is borne by the patient, by the influence of the diet on the progress of the disease, and by the complete evacuation of the food before the expiration of seven hours. Leube, in the determination of the digestibility of food in disease, began with patients who were able to comfortably digest but little food within the period of seven hours. Beginning with these few articles of most easily digested food, a table of decreasing digestibility was made by studying cases of disease of less and less severity. Consequently, Leube includes in the conception of digestibility in disease not only the duration of the sojourn of a food in the stomach, but also the sensations which it excited and the influence which it exerted on the progress of the disease. The stomach-tube was used to determine whether the stomach was empty at the expiration of the arbitrary seven hours. The following progressive diet of Leube is extensively prescribed, and has proved very valuable:

I. Bouillon; Leube-Rosenthal meat solution; milk; soft-boiled or raw eggs; dry toast or crackers; water or natural indifferent effervescent (CO_2) water.

II. Boiled calf's brain; boiled sweetbread (thymus of calf); boiled young chicken; boiled squab; cereal soups; tapioca cooked in milk; boiled calf's feet.

III. Sirloin pulp steak; grilled sirloin steak; scraped raw ham; mashed potatoes, baked, with a little milk and butter; a little white bread; try coffee or tea with milk.

IV. Roast beef; roast chicken, venison, partridge, and veal; boiled lean fish; macaroni; bouillon or rice soup; spinach; a little wine.

V. Baked apple; all common foods; finally, salads, vegetables, and stewed fruits.

The method of Leube was improved and employed by Penzoldt, but under better control and under precautions against error. The results are comprehensive and valuable, but were obtained by the study of digestion in health. The stomach-tube was used to determine the progress of digestion and the exact moment when the stomach is completely empty after

the eating of a definite quantity of a particular food. The table of Penzoldt, which in its chief articles is as follows, is a table of digestibility in health. The time represents the period within which the food leaves the stomach. The quantity is given in grams:

One to Two Hours.	Two to Three Hours. (*Continued.*)	Three to Four Hours. (*Continued.*)
100–200 water.	150 cauliflower (boiled or salad).	100 beefsteak pulp.
200 tea, " coffee, " cocoa } plain.	150 asparagus (boiled).	" roast beef.
" beer.	" potatoes (mashed).	200 Rhine salmon.
" light wine.	" cherry compote.	70 caviar.
100–200 sterilized milk.	" raw cherries.	150 black bread.
200 meat tea (pure).	20 white bread.	" white bread.
100 soft-boiled egg.	" zwieback.	100 Albert biscuits.
	50 Albert biscuits.	150 boiled rice.
		" " spinach.

Two to Three Hours.	Three to Four Hours.	Four to Five Hours.
200 coffee with cream.	230 stewed young chicken.	210 roast pigeon.
" cocoa with milk.	230 partridge, broiled.	250 " fillet.
300–500 water.	240 stewed pigeon.	" beefsteak, grilled.
" " beer.	195 roast "	" smoked tongue.
" " boiled milk.	250 beef (raw or boiled).	" hare.
100 raw egg or omelet.	" calf's foot, boiled.	240 roast partridge.
250 calf's brain, boiled.	160 ham boiled.	250 " goose.
" sweetbread, boiled.	100 roast veal.	280 " duck.
70 raw oysters.	" beefsteak.	150 mashed lentils.
200 boiled carp.		200 mashed garden peas.
" " pike.		150 snap beans.

Penzoldt has constructed a progressive diet which is based on the digestibility of food by the normal stomach and which agrees in its main features with that of Leube, which has already been given. Each diet consists of a combination of various foods and demands more and more work of the stomach.

First Diet.—Cup of lean beef broth without salt; glass of sterilized milk, pure or with one-third lime-water; one or two boiled eggs, or one or two raw eggs stirred in hot meat broth; 30 to 40 gm. of Leube-Rosenthal meat solution; 6 crackers (Albert); ½ of a glass of plain or charged table water.

Second Diet.—One hundred gm. of boiled calf's brains; 100 gm. of boiled sweetbread (thymus gland of calf); 1 boiled squab; 1 boiled chicken (partridge size); 100 gm. of raw scraped or chopped beef with crackers; 30 gm. of tapioca in milk. The meats should be freed from fat and fibrous tissue, and had better be stirred in meat broth before serving.

Third Diet.—One pigeon broiled with fresh butter; one broiled chicken; 100 gm. of rare roast beef; 100 gm. of scraped

raw ham; 50 gm. of zwieback or French roll; 50 gm. of mashed or mealy potatoes; 50 gm. of cauliflower (bloom) cooked in salt water.

Fourth Diet—One hundred gm. of roast venison; 1 roast partridge; 100 gm. of rare roast beef (hot or cold); 100 gm. of roast veal cutlet; 100 gm. of boiled lean, fresh fish; 50 gm. of Russian caviar; 50 gm. of thoroughly cooked rice gruel; 50 gm. of boiled asparagus (tips); 2 scrambled eggs with a little fresh butter and salt or omelette soufflé; 50 gm. of sauce of stewed fruit; 100 gm. of warm claret (Bordeaux).

A table of digestibility to be of clinical value must rest on a broader basis. Not the influence of the food on the motor function and on the other functions of the stomach in health, but the ease and surety of the useful and unconscious transformation of food into assimilable nutriment by the digestive tube is the clinical measure of digestibility. This clinical conception includes the following four elements:

1. The food must be easily soluble in the digestive juices.

2. Its physiological action must be such as is most favorable to its digestion and absorption, and such as exerts a remedial influence by favoring or exciting the diseased part.

3. It must not be liable to undergo fermentation or decomposition before it reaches the point of digestion and absorption.

4. Its digestive products must be easily and quickly absorbed after their formation, or must resist superdigestion and destruction by micro-organisms.

5. The digestion must be accomplished without pain or discomfort.

From a clinical point of view, the digestibility of a food is not absolute, but individual and variable. The food and its palatable properties are the only fixed elements in the problem. The stomach and the intestines are as variable in power as is the general strength of men, and are as individual as the human face. Often touched by hereditary taint, on both the stomach and intestines are written the biography of their possessor, the record of past diseases, and the salient points in the mode of life. Both become adapted to their environment, which is composed, on the one hand, of the matter in contact with the mucous membrane, and, on the other, of the state and characteristics of the organism. The food and its associations, the products of digestion, fermentation, and decomposition, the flora of the stomach and intestines, are, for no two persons, exactly the same. Secretion is variable, movement is variable, and all of these are individual. Con-

sequently, individualization is a commanding principle of dietetics and a modifying factor of digestibility. A knowledge of food, of its general digestibility, of the state and needs of the organism and of its parts, can serve only as guiding threads. The course is zigzag, but the variations in health are limited, and the variations in disease are quite constant. An average or sweeping curve, however, can be marked out as a clinical guide.

From this point of view the chief foods of interest to the physician in the treatment of the diseases of the stomach will be studied in their physiological action, the degree of resistance which they offer to digestive transformation, and their liability to undergo destructive or injurious changes; and in this study must be included the action on the stomach, both in health and in disease.

Meats.—The physiological action of the meats is dependent on their mechanical and chemical influences, and on the associations of the albumin with the other alimentary principles and with the products which appeal to the palate. Consequently, their physiological action is determined by the following factors:

1. Physical state.
2. The preparation for the market.
3. The preparation for the table.
4. The associated products, of which the chief one is fat.
5. The quantity eaten.

Meat is the natural stimulant of gastric secretion, an adaptation, through use, of the activity of a part to the special work which it is intended to perform. The means and the end are fitted to each other, and special opportunity excites purposive activity. The chemical work of the gastric secretion is expended on the albumins, and in the ordinary diet these consist chiefly and most universally of the meats. Meat is thus the chief natural excitant of gastric secretion, and as secretion is so greatly influenced by the movements of the stomach, in the course of nature normal and suitable muscular action has become associated with the natural secretion. So, likewise, with the other functions of the organ. The meats, then, excite the stomach to the fullest normal activity. The process and evolution of the digestion of meat represent fully and truthfully the functional work of the stomach, provided the meat is palatable to the individual and does not break too rudely his diet habits. Here, again, the personal element enters and destroys exactness in the detailed ordering of a diet. The physician is the judge, and it is his duty to

apply the general laws and precedent to the individual case, under the guidance of experience.

To represent the comparative evolution of the digestion of meat and of bread and milk, a piece of the round of a properly-fattened two-year-old steer, after hanging, may be selected, the fat and fibrous tissue cut away, and the pulp, separated by a non-cutting chopper, made into masses about an inch thick and two inches in diameter and quickly cooked on a hot griddle so as to leave the center rose-red. Two of these may be taken with a cup of weak tea.

Comparatively, the gastric secretion is more intense and the evolution of digestion is longer than with either milk or the ordinary roll test-breakfast. The digestion of a glass of milk is near its end at one and a half hours, that of bread at two hours, but that of the beef pulp at three and one-half hours.

The total hydrochloric secretion is greater with the meat, but it is sufficient and not excessive. At the height of digestion there is always a trace of free HCl, but only a trace. The HCl is utilized, but there is no excessive secretion, as with the digestion of bread, and the trace of free HCl continues to the end.

The evolution of the digestion of a beefsteak or roast beef is not the same as that of the beef pulp. The excitation is more intense; less hydrochloric acid is combined, even if the analysis be made on the unfiltered contents; more hydrochloric acid remains free; the duration of digestion is prolonged; and between the third and fourth hours the quantity of digestive products is greater. The difference is due to the presence of fat and of fibrous tissue, to the larger particles, entailing more intense excitation, to diminished utilization of the secreted HCl, to accumulation of stimulating digestive products, and to delayed evacuation.

The action of the meats on secretion is dependent also on the quantity which is eaten, for the greater the quantity the longer the sojourn in the stomach. In this respect the table of Penzoldt gives very exact and valuable information. Some of the meats are digested much more slowly and others more rapidly than the beef pulp. The greater the quantity of fat and the less minutely divided the meat, the longer is its sojourn, the slower its digestion, and the more out of proportion is the quantity of secretion to that which is required for its transformation. Young chicken, veal, calf's brain, squab, and sweetbread are more digestible when boiled, while the older and red meats are better borne when grilled or roasted. The meats are among the best utilized foods. In health, only

from three to five per cent. of the total quantity of albumin ingested is unabsorbed; little is destroyed in the digestive tube and little is eliminated in the feces. But this is true only while the functions of the intestines and pancreas remain normal. To excite these functions physiologically and to maintain the balance of nutrition, other kinds of food should be combined with the meats. The action on intestinal peristalsis is altogether insufficient, but the decomposition products may produce irritation and putrefaction if favored by the administration of meats exclusively and in larger quantity than in the normal diet.

Meat furnishes one of the best means of supplying the nitrogenous needs of the organism, but should not be selected when the object is to give the stomach physiological rest. Its gastric digestion is an example of energetic purposive work without waste. In all diseases of the stomach accompanied by a diminution of the hydrochloric acid secreted, the quantity of meats in the diet should be reduced to a minimum. The white meats are preferable when it is desired to favor the stomach, because they contain less extractive matter, less hemoglobin and iron, and, when of the proper kind, also less fat. The red meats are to be preferred whenever intestinal putrefaction exists and whenever it is desired to stimulate the stomach gently and to exercise it.

Where there is excessive hydrochloric secretion the meats should be given in increased quantity, in order to combine the acid and to secure for the organism the advantages of the digestive and secretory disorder. Here it seems to make little difference chemically whether the best of the white or the red meats be given, so long as both are very finely divided.

In the dynamic affections of the stomach which are not accompanied by a disorder of secretion, the meats, if properly selected, are usually borne. Some cases of morbid sensibility of the stomach form an exception to the rule. The meats, like all other foods which act energetically on the stomach, should not be prescribed in the anemias associated with vascular disturbances. Their digestion is then often accompanied by a very rapid pulse, and sometimes excites a *bruit de galop*, or reduplication of the second sound of the heart, or cardiovascular dyspnea. Here a milk diet, if there be no contraindication, combined with rest in bed, is most suitable.

Gastric putrefaction is very rare, except it occur as an accident; but intestinal putrefaction is quite frequent, and it may be necessary temporarily to withhold all meats in order to control this condition. Gastric fermentation is common

enough, and is often accompanied by intestinal fermentation. An exclusive but temporary meat diet (with, when possible, a little dry toast and lemon-juice) is then by far the best treatment. Some little temporary injury to nutrition may be done thereby, and the emaciation should not be allowed to go too far; but cases of this kind are not rare where no other diet has any permanent or curative effect. Carcinoma is a formal contraindication to an exclusive meat diet. When using meats in the dietetic treatment of diseases of the stomach, lean meat should be selected and the fat should be carefully removed. The fat alters the digestibility and the physiological action of the muscle pulp, prolonging digestion and increasing excitation.

The most digestible meats are beef, mutton, fowl, some kinds of game (grouse, pheasant, quail, venison), calf's sweetbread, and calf's brain. Goose and duck are too fat, and stay in the stomach about five hours. Pork should never be permitted, but lean boiled ham is often well borne. Roast and broiled chicken and squab are digested in about three and one-half hours. The young meats, and brain, and sweetbread, should not be roasted or grilled. Raw meat is no more easily digested than when it is cooked and finely divided.

The commercial preparations of meat are very numerous, and their extensive manufacture is sufficient evidence of their great consumption. Convenience and concentration are their chief advantages. Some of them, when fresh, are valuable both as food and as remedies. The use of most of them in the treatment of the diseases of the stomach can be easily dispensed with if a good cook is at command, and some of them are veritable irritants and purgatives.

The commercial albuminous preparations which contain large percentages of mineral and extractive matter excite gastric secretion and irritate the mucous membrane of the alimentary tract. Their transformed albumins easily putrefy in the bowels, and are no better utilized than the albumins of the ordinary foods when in solution or in a state of fine division. They possess no greater nutritive value than is represented by the caloric value of the alimentary principles which they contain, and they can be administered only in very small quantity without disordering digestion. The following table shows the nutritive value of some of the prepared foods in comparison with that of expressed beef juice, meat powder, eggs, milk, and beef:

100 GM.	SOLUBLE ALBUMIN.	EXTRACTIVE MATTER.	MINERAL MATTER.	CALORIC VALUE.
Valentine's meat juice,	6.7	22.8	11.4	27.
Liebig's meat extract,	20.5	38.	23.	84.
Johnston's beef extract,	36.7	8.	9.	150.
Somatose,	80.	0.	6.7	320.
Nutrose,	90.	0.	0.	369.
Eucasin,	85.	0.	0.	348.
Expressed beef juice,	3.	3.	1.5	12.
Meat powder,	69.5	. . .	13.	285.
Egg (white),	. . .	. . .	. . .	53.
One egg,	. . .	. . .	. . .	70.
Milk,	. . .	. . .	. . .	70.
Beefsteak,	. . .	. . .	. . .	133.

Somatose, nutrose, and eucasin are excellent preparations, but not more than 50 gm. of either can be administered during the twenty-four hours without disordering digestion. Consequently, little more than one-half of the nitrogenous needs of the body can be supplied by these foods, and they furnish only about one-fifteenth of the quantity of nutriment which is required daily.

Bouillon, meat broths, meat teas, expressed meat juice, meat powder, etc., have their special uses and action on the stomach. The indications for them in the particular diseases will be given in Sections IV and V.

Fish.—On account of the high percentage of fat, few fish can be employed in the treatment of the diseases of the stomach; but when these few can be obtained fresh, a very delicate, digestible, and valuable albuminous food is at our command. The lean, fine-meated, perfectly fresh and boiled or baked fish are the only varieties permissible. Among these the following may be named: sole, weakfish, bass, trout, German carp, chicken halibut, flounder, sheepshead, pike, the lean parts of bluefish and of shad.

The action of lean fish on the stomach is much less energetic than that of meat. Secretion is not so active, and when fish is eaten alone is hardly sufficient for its digestion. In this respect it more closely resembles the white than the red meats. Digestive transformation is quite rapid, and the stomach is empty in about two hours after a breakfast of a small piece of sole, a little lemon-juice, and a cup of tea.

The albumin of the lean fish is well utilized, only about two per cent. being lost. About ten per cent. of the fat, however, is recovered in the stools.

Fish of the kind recommended may be prescribed to give

a little variety to the diet, and in larger quantity where meats, on account of their comparatively high price, can not be secured. It is a food which is useful in favoring the stomach, but it can not be employed as a physiological remedy where it is desirable to stimulate secretion.

Milk.—Milk is one of the most widely used and important foods. The only complete and natural food in infancy, in later life milk and its products are also largely consumed.

König gives as a daily average consumption for each individual, ¼ of a liter of milk, ten gm. of butter, and ten gm. of cheese, statistics based on the consumption in large cities. In small towns and in the country the consumption is notably greater, and may be attributed to its cheapness, nutritive value, palatableness, and easy digestibility in health. Its fluid form and its eliminating properties have won for it a very general use in therapeutics.

From a clinical point of view, this complete fluid food is not without its imperfections. The subjective symptoms following its use may to some persons be so disagreeable and peculiar as to reveal a so-called idiosyncrasy, and may be so pronounced as to preclude its use. It also forms a good culture fluid for many germs, under whose influence it rapidly undergoes destructive changes.

The figures of Freudenreich demonstrate the high value of milk as a culture soil, and the influence of the temperature on germ growth. On arrival in the laboratory, one c.c. contained 9300 germs:

	At 15° C.	At 25° C.	At 35° C.
Three hours later, ...	10,000	18,000	39,000
Six hours later, ...	25,000	172,000	12,000,000
Twenty-four hours later, .	57,000,000	577,000,000	50,000,000

The nutritive value is not only markedly diminished by these germs, but the products of germ activity are injurious and modify its physiological action. The most common fermentation products are lactic and butyric acids, and tyrotoxicon is formed by its putrefaction. The germs of fermentation and putrefaction may be introduced into the body with it, or may be found awaiting its arrival in the stomach or intestines. The first imperfection may be partly remedied by sterilization when fresh, and this procedure is always advisable in order to avoid the introduction of pathogenic bacteria. Sterilization may be done in the ordinary way with an apparatus like that of Soxhelet. But a better plan is pasteurization. The milk is kept at a temperature of 70° C. for five

minutes, then rapidly cooled by placing it in the ice-box. It is then kept lukewarm for about two hours, and again heated to 70° C. The disadvantages of a high temperature are thus avoided, and the resistant spores are germinated and killed, whereas by the ordinary method the spores are usually not killed. The milk should be sterilized or pasteurized immediately after the milking, while it is perfectly sweet; or in hot weather it should be kept cold until the procedure can be employed.

For the adult, another disadvantage is its small nutritive value in comparison with its total mass. To supply the number of heat units represented by the biological coefficient for the adult of average weight, about four liters are required daily. In any other condition than rest the balance of nutrition can not be maintained at the normal level on an absolute milk diet, and its exclusive use by the adult constitutes a starvation-cure.

The average composition of milk is:

	Casein and Albumin.	Fat.	Milk-sugar.
Durham and Ayrshire,	3.4	3.5	5.5
Jersey, ...	3.3	4.2	5.7
Guernsey, ...	4.0	5.1	4.4
Mixed, ...	3.5	3.7	4.9
Four liters, ...	140 0	148.0	196.0
Repose ration, . . .	100.0	50.0	400.0 (carbohydrates)
Three liters, ...	105.0	111.0	147.0

Theoretically, four liters daily should maintain the nutrition of the adult; but a large proportion of the milk-sugar may be lost in disease, and about five per cent. of the fat and ten per cent. of the casein are unabsorbed. The milk is markedly diuretic, and increases, when given alone, the urea elimination about one-third (Chibret). An exclusive milk diet, except when the patient is kept warm in bed, produces a marked loss of strength, though the body may lose little weight.

As an ideal food, its combination of the alimentary principles is defective. The carbohydrates are deficient and the fat is excessive; and until it be proven that the form of albumin is of no consequence, its almost exclusive casein may be considered a defect.

Yet another imperfection is in its digestion and utilization. Contrary to the general belief, a large percentage of its alimentary principles, when given as an exclusive diet, reappear in the stools or are lost by fermentation.

The following table of average percentages shows this loss and its increase with the quantity daily administered:

	Casein.	Butter.
Two liters, . .	5 per cent. in stools.	3 per cent. in stools.
Three liters, .	8 " " " "	5 " " " "
Four liters, . .	12 " " " "	6 " " " "

The percentage of loss in disease may be greater, and the milk-sugar (none of which is found in the stools) may serve entirely as food for bacteria. The caloric value of the casein and the butter which are utilized in three liters is 1450 Cal.

One feature in its digestion is clinically of very great importance—the products formed by its coagulation by organic and mineral acids, and by the labferment, differ materially in their digestibility and in their physical and chemical properties. The acids form a clot which is the same as clabber. The casein is split by the labferment into two products, one of which is casein-albumin and is held in solution by the phosphate of lime, and the other is precipitated in combination with calcium as a base, and is digested in the intestines. It is practically important that the curdling be produced by the labferment and not by an acid, as is often the case when the stomach is diseased.

The physiological action of milk on the healthy stomach is very slight. With a glass of milk the height of secretion occurs in about forty minutes, and at the expiration of one and one-half hours the stomach is empty. During its digestion there is no free hydrochloric acid, or the merest trace of it. The HCl in organic combination reaches its height sooner than with the test-breakfast, and the acidity due to it is commonly a fraction greater. As compared with the digestion of the test-breakfast the digestion of milk is more rapid and the acid is more rapidly and completely utilized. If a large quantity of milk be taken the total acidity increases, and there is often an appreciable quantity of lactic acid, because the sojourn in the stomach has been longer.

1. Gastric excitation is less than with bread.

2. The hydrochloric acid is completely and rapidly combined.

3. In small quantity it is evacuated in a short time; and there is no sign of irritation left, such as a tendency of secretion to continue.

4. In large quantity the secretion of hydrochloric acid is insufficient, lactic acid is formed and inhibits the rapid evacuation of the chyme. If a notable quantity of lactic acid be

present, the stomach often "splashes" about two hours after the beginning of digestion.

The physiological action of milk on the diseased stomach is variable. In excessive secretion with the motor function intact it acts as a sedative, relieving irritation; but in diminished secretion and in motor insufficiency, whatever be the state of secretion, it undergoes imperfect digestion and ferments, producing sometimes butyric and sometimes lactic acid. Butyric acid formation is frequent when the hydrochloric acid is secreted in excess. These acids produce great irritation and delay the evacuation of the contents of the stomach.

When milk is perfectly digested it exerts little action on the intestines; the utilization is almost complete; there is no excessive flatulency, the urotoxic coefficient is markedly diminished, and there is less than the normal quantity of indican found in the urine after the healthy digestion of a mixed meal. The lower half of the bowels is given almost complete rest. The action on the motor function is insufficient, and constipation is a very troublesome result.

But the intestinal digestion of milk is not always so perfect. The lactose may ferment; the lactic acid formed is irritating and constipating, or the butyric acid is even more irritating and is likely to produce catarrh with sometimes diarrhea and an intense headache. The bowel may be distended with gas, which is chiefly hydrogen. Even when the milk undergoes these changes and produces these results, the urotoxic coefficient remains constantly low. If fermentation becomes active with an exclusive milk diet, inanition is rapid. The water, the lactose, and some of the germ products are actively diuretic, and the urea eliminated is increased. The loss of nutritive equilibrium produces increasing loss of strength.

A very serious objection to the employment of an exclusive milk diet is the almost constant activity of the stomach as a consequence of its frequent administration. An exclusive milk diet, when digested, demands no very great work of the stomach at one time, but it gives the organ no rest.

The indications and contraindications to the use of a milk diet may now be formulated. The diet is indicated, not when it is possible, but when it is better than any other.

The milk diet is valuable in the treatment of ulcer of the stomach. Indeed, when in ulcer the motor function is normal and milk does not produce intestinal indigestion, an exclusive milk diet is the sovereign remedy, combining the excessive hydrochloric acid secreted and making little

demand on the functions of the stomach. But it does not give the stomach functional rest, and if there be gastric stagnation or intestinal fermentation or morbid sensibility of the duodenum, it will have to be discarded in favor of another diet.

Milk is also the best food in the irritative stage of acute gastritis, provided it is borne well by the intestines. But it rarely agrees in acute gastritis when the contents are neutral or very slightly acid or when there is fermentation.

A milk diet acts most happily in adenohypersthenia gastrica associated with intestinal putrefaction and an excessive quantity of indican in the urine. In this special condition a milk diet is remarkable in its effects, soothing the irritability of the gastric glands, arresting intestinal putrefaction, reducing the congested liver, and relieving the auto-intoxication by free diuresis.

A milk diet is contraindicated in all the diseases of the stomach expressed functionally by a notable diminution in the quantity of hydrochloric acid secreted. Consequently, in all cases of chronic asthenic gastritis, and in many cases of acute gastritis, milk should be prohibited. It is the worst possible food in carcinoma with retention. Milk should be prohibited in all the diseases of the stomach accompanied by retention. In some forms of stagnation it may be proper and valuable if it agrees well with the intestines. The morbid glandular irritability which often accompanies myasthenic stagnation may be relieved by a milk diet. If the myasthenic stagnation be relative, the stomach is often strong enough to evacuate a glass of milk within the normal period, but becomes insufficient when a larger meal is given. Milk may be made the basis of the liquid diet appropriate in obstructive stagnation. A milk diet is often curative in prolonged digestion due to excessive secretion, but in all forms of motor insufficiency milk must be forbidden whenever it undergoes or produces fermentation.

But even when suited to the stomach, a milk diet may be contraindicated by its action on the intestines and on nutrition. When the bowel is sensitive in its upper third, or is myasthenic, or is the theater of active fermentation, a milk diet should not be prescribed. Harm may also be done by prescribing a milk diet when the patient is already weak and emaciated as a result of disease of the digestive tube.

There is no diet that requires more careful watching and selection than one of milk. Its indiscriminate and routine employment in the diseases of the stomach gives, in the suit-

able cases, most excellent results, but in the remaining cases either does no good or produces serious injury.

Preparations of Milk.—*Kefyr* (so-called koumiss of commerce).—The most valuable preparation of milk in the treatment of diseases of the stomach is kefyr, which is the product of special fermentation of cow's milk. It is a household drink in the Russian Caucasus, and is claimed to be a gift from Mohammed to the people.

The kefyr ferment in the dry state preserves its activity unimpaired for one or two years, and is sold by the Russian pharmacists, often adulterated, at a high price in the form of yellowish-white, dry, brittle granules. Kefyr contains essentially two germs, one of which, the dispora caucasica of Kern, converts milk-sugar into lactic acid and partly into an isomeric form which is easily transformed by the other yeast-plant (saccharomyces cerevisiæ) into alcohol and carbonic acid. Other ferments and bacilli have nothing to do with the formation of the characteristic product, but often have to be picked out from the kefyr ferment or killed by a weak solution of salicylic acid. The writers who have studied the process advise a thorough cleansing of the ferment, after each use, by stirring in a one per cent. solution of soda for a few hours, picking out the slimy grains, and leaving the ferment for twenty-four hours in a 1 : 5000 solution of salicylic acid in water. The preparation of kefyr, as described by Biel, consists of two distinct parts—the preparation of the ferment and the transformation of the milk.

The dry ferment is soaked for about half an hour in water at 30° to 35° C., and the water is then poured off and replaced by fresh water at 20° C., in which the yeast is left for twenty-four hours. The white ferment is next washed on a sieve with warm water, and then added to a liter of sweet sterilized milk, in which it is left and occasionally shaken for twenty-four hours, when the cleansing is repeated and new sterilized milk again used as the culture fluid. After ten or twelve days of this treatment the yeast rises to the surface of the milk on account of the fermentation excited, the cheesy odor is no longer present, and the milk shows a slight coagulum at the end of twenty-four hours. The yeast is now ready for use in the preparation of the kefyr.

A glassful of the prepared yeast is added to a glass vessel containing a quart of fresh sterilized and cool milk, corked with clean cotton, and set aside in a light, airy room at a temperature of about 20° C.

After about twenty-four hours, during which the vessel,

without uncovering, has been frequently shaken, the milk becomes creamy and has a sharp and sweet taste. The preparation is now strained and bottled. A champagne bottle is filled about one-third full with the prepared milk, and enough sweet sterilized milk (preferably skimmed milk, as the fat is liable to be decomposed) is added to almost fill the bottle, which is then corked, fastened, and stationed, top down, in a room at 15° C. The bottles should be shaken every two or three hours, but never uncorked. The alcoholic fermentation should continue three or four days, and when it stops the bottles should be kept on ice. For medicinal purposes it is not advisable to use the kefyr until the fermentation has spontaneously stopped and a lasting foam forms on shaking.

It is more palatable than koumiss or matzoon, should be more generally used than it now is in the United States, and when well prepared has the following average composition:

Kefyr.	Casein.	Syntonin and Albumoses.	Butter.	Milk-sugar.	Lactic Acid.	Alcohol.
100	2.5	1	2–3.5	2	1	1–1.5

Physiological Action.—The physiological action of kefyr is very different from that of milk, and this difference is due to the fact that it contains, in addition to the usual ingredients of milk, a notable quantity of alcohol, lactic acid, and the kefyr bacillus and yeast. In virtue of these ingredients the excitation is more pronounced than that of milk, the gentle action of which both on the secretory and motor functions of the stomach is so well known. As compared with milk, the evolution of secretion is more rapid but more prolonged, and the curve of acidity rises higher. The total quantity of HCl secreted is greater, and a moderate quantity of free HCl is present during the second hour. The stomach seems to empty itself more slowly. At the end of two hours ordinarily a small quantity of contents can be withdrawn, the hydrochloric acidity of which is still high. In a word, the secretory excitation is more intense and more persistent than with sterilized milk, and its digestive transformation is much more rapid and complete.

The physiological action on the intestines is not marked in health. Kefyr is better utilized than milk, and only a very small percentage is found in the stools. The small percentage of alcohol promotes both gastric and intestinal absorption.

The very small and infrequent stools reveal its inactivity on intestinal secretion and movements. It constipates, but rests the lower part of the intestines.

Clinically, the contraindications to the use of kefyr are formal. In all forms of irritative gastric trouble it should not be prescribed. It gives temporary relief in adenohypersthenia gastrica, as all albuminoid foods do; but the excessive secretion of hydrochloric acid is either increased or not controlled after long use. Alone, it is capable of producing a catarrh in this disease, when both the total acidity and that due to free HCl may disappear, or the partial neutralization by the inflammatory exudate may lead to an erroneous and too favorable opinion. In stagnation with fermentation it is injurious. In hyperesthesia of the gastric mucous membrane the sensations of the patient will soon call the attention of the physician to the increased discomfort. It is a valuable remedy in all intestinal diseases except myasthenia.

In acute or subacute alcoholic gastritis, kefyr is most valuable. It is especially useful after a debauch. In case there exist no forced contraindication to its use, on account of an associated condition, its employment in acute alcoholic gastritis during one to three days gives very remarkable results and is much better in this disease than ordinary milk.

Another condition in which it may be used with benefit is in subacidity, whether associated or not with a lesion of the mucous membrane. It often acts very happily in these cases, provided there is neither myasthenia nor morbid sensibility of the mucous membrane. The treatment may be begun and continued for three or four days with kefyr alone.

Again, in diarrhea, accompanied by fermentation and irritation, associated with organic or functional gastric subacidity, kefyr may at times be used with advantage. The results are more satisfactory when the diarrhea is due to putrefaction. As a food, it is easily digested and rapidly absorbed in the stomach and upper part of the small intestine. As a remedy, it acts chiefly by suddenly changing the culture soil of the germs to the action of which the persistence of the diarrhea is due. To change the flora of the intestines it should be used as an exclusive diet for a few days.

Kefyr was introduced as a means of inducing superalimentation in the treatment of tuberculosis. In the Russian establishments it is given as a supplement of a richer diet in a quantity varying from one to six liters a day, as tolerance becomes established. The quantity for the day is divided into three parts: One portion is taken before the meat-breakfast. The second portion is begun half an hour after breakfast and finished an hour before dinner. The third portion is begun two hours after dinner and finished an hour before supper.

It is to be taken slowly in sips, a short promenade preceding each glass. Given according to this method, it may be used with advantage as a supplementary food in affections of the stomach when there is no atony of the muscular layer. During the cure no other fluid should be permitted. The results of its use as a surplus food in the gastric troubles of phthisis are most excellent.

Koumiss.—Koumiss is a preparation of milk similar to kefyr, but, properly speaking, is made out of mare's milk. It is a drink of the ancient Scythians, and is very popular in southern Russia, whence its use as a remedy and a food has spread over the civilized world. It is valuable on account of its easy digestibility. Koumiss, outside of Russia and Siberia, is made of cow's milk through a double lactic acid and alcoholic fermentation. There may be little difference between koumiss and kefyr, but koumiss is properly made from a combination of ferments, and not from a pure culture, as is kefyr. An additional quantity of milk-sugar is often used—or cane-sugar with maltose may be used in its preparation by the yeast of beer—and it is ripened at a lower temperature. Consequently, the preparation is less constant, often too acid or incompletely fermented, and the associated germs are very variable and not unimportant in the possible modifications of its physiological action. Kefyr is preferable unless the koumiss be prepared in a like manner and given the wrong name in commerce.

The third-day koumiss, made of cow's milk with added sugar, which is the form used in diseases of the stomach, has the following average composition:

	Casein.	Syntonin and Albumoses.	Butter.	Milk-sugar.	Lactic Acid.	Alcohol.
Unskimmed milk, . .	2	1.5	3–5	3	1	1.5–3
Skimmed milk, . . .	2	1.5		3	1	1.5–3

The quantity of fat contained in milk seldom constitutes an objection to its use as a remedy. Its presence interferes little with the chemical changes which milk undergoes in the stomach, but fat in large quantity, like all sweets, diminishes the already slight action of milk on the muscular layer, or, like the fatty acids, if decomposed produces an intense congestion of the mucous membrane.

The fat rises to the top because it is lighter, and not on account of any change in the milk. But the cream also contains casein, albumin, sugar, and salts, which the fat-globules have carried along with them. The upper layer, however, in

which the larger fat-globules collect, is not so rich in these nutritive ingredients.

The method used in skimming the milk and obtaining the cream is important. The milk, after being drawn in a clean place, by clean hands, from clean udders, into clean vessels, is immediately sterilized by the spore-killing process and kept for twenty-four, forty-eight, or seventy-two hours at a temperature of 10° to 15° C. The cream can then be poured or siphoned off just before drinking. The skimmed milk is in this way perfectly sweet and sterilized. When the machine can be obtained, the milk before sterilization may be separated by the aid of a centrifugal apparatus. The skimmed milk obtained in this way is too poor in fat, as much as possible of which should be introduced with this already insufficient food. It is well established that the emulsionized fat in milk is never contraindicated by a disease of the stomach when milk-sugar can be given. The effort to exclude the fat of milk in the treatment of the diseases of the stomach is a mistake. Consequently, no trial should be made of skimmed milk when sweet sterilized milk is not well borne. Also buttermilk, clabber, whey, and peptonized milk, and all the sweet condensed preparations, in our experience are valueless in the treatment of the diseases of the stomach.

The cream separated from the milk in the preparation of skimmed milk is a very rich, digestible, fatty food, the use of which may be advisable during the course of the treatment of some of the diseases of the stomach when a concentrated diet must be ordered. Its richness in nutriment is variable.

Eggs.—The egg, a secretive and formative product of the reproductive organs of the female bird, is a very valuable and widely-used aliment in both health and disease. The composition of the eggs of all birds is nearly the same.

The egg of the hen varies in weight between 45 and 70 gm., and is equal in nutritive value to about 40 gm. of meat, and contains about the same quantity of albumin and fat as 150 c.c. of milk. It is often considered a complete food, but it is no more so than fat meat. The physiological action is modified by the preparation, and that of the white alone is very different from that of the whole egg.

The hard-boiled white of egg rapidly excites secretion, and the height of digestion falls between thirty and forty-five minutes, when the quantity of combined and free HCl is greater than that of the test-breakfast. In seventy-five minutes after eating the white of one egg the stomach is empty, secretion promptly ceases, and the products of digestion do not

accumulate. If the hard-boiled egg be very finely divided before it is eaten, secretion is less rapid and less intense, and the free HCl is less than when the egg is simply masticated. The stomach is empty in one hour and enters at once into repose. The digestion of egg-water is like that of the pulverized white of egg.

The digestion of the whole soft-boiled or poached egg is modified by the increase of fat and of salts. Secretion is slower in its evolution, digestive products are more plentiful, the curve of free HCl is slower in its descent, and the stomach is not empty until near the end of two hours.

The percentage of utilization in health is very high. Only about three per cent. of the albumin and five per cent. of the fat escape absorption. Both the white and the yolk putrefy much more readily than meat.

The whole egg constitutes a very fat food, and very much of the yolk is unsuitable in myasthenia. The white should be lightly coagulated or in solution when rapidity of digestion is an object and excessive excitation is to be avoided. The white agrees well in adenohypersthenia, and also in myasthenia, but in both of these functional states much of the yolk is likely to do harm. In gastric retention and intestinal putrefaction eggs should be excluded from the diet. In some persons one egg may be sufficient to initiate intestinal putrefaction, and to produce a few colicky movements and an abundant formation of hydrosulphuric acid gas. Egg stirred in bouillon just hot enough to coagulate the white is as easily digested as the soft-boiled egg, but the salts in the bouillon slightly increase the hydrochloric acidity. A soft-boiled egg is digested in less than two hours, a raw or a scrambled egg in two and a half hours, but a fried egg or an omelette requires nearly three hours.

Cereals.—The cereals are very variable in their digestibility and action on the digestive tube. Practically, only a few of them are useful in the treatment of the diseases of the stomach, but these few, on account of the digestible starch which they contain, possess a unique and commanding value. Of these, the preparations of wheat, rice, hominy, and oatmeal are the most important.

The physiological action of common bread on the healthy stomach is well known, forming the only solid constituent of the Ewald-Boas test-breakfast. The action on secretion is somewhat in excess of the requirements of its digestion. At the end of half an hour, traces of free HCl may he found unutilized, and at the expiration of one hour this

free hydrochloric acidity is increased to a considerable quantity, and is represented by a decinormal acidity of 10 to 15, which persists until the stomach is empty. The evacuation of the stomach is complete in about two hours, and there is no great accumulation of digestive products. The motor function is sufficiently stimulated.

The intestinal digestibility and action are very different from those of any of the foods which have been thus far considered. A very large percentage of the albumin and fat is unabsorbed. A much greater quantity than of the meats or fish or milk or eggs is used in feces formation, and intestinal peristalsis is more regular and efficient. This action on the movements of the bowels is an advantage, but the loss of unabsorbed nutriment is very great. From 1 to 20 per cent. of the albumin and nearly half of the small quantity of fat are recoverable in the stools. The starch is utilized to within one per cent. in health, and though in disease the quantity found in the stools is not much increased, the clinical guides often show a large loss by fermentation.

In disease, the physiological action of bread on the digestive tube is remarkably modified if the starch undergoes fermentation. The lactic, butyric, and other acids formed may produce extreme irritation. The starch itself, in excessive secretion, is incompletely digested by the saliva, and often increases the glandular irritation and activity.

The bread used in the treatment of the diseases of the stomach should be made of very finely-ground white flour. With the whole wheat bread the utilization is much lower, and the gastric excitation in health somewhat greater than with white bread. About 30 per cent. of the albumin of the whole wheat bread is recoverable in the stools, and from five to ten per cent. of the starch escapes absorption and fermentation. Clinical experience and the use of the clinical guides prove that the crust of bread or dry toast browned through and through is well borne in the diseases of the stomach.

The wheaten grits are, when thoroughly cooked, suitable for the treatment of the diseases of the stomach. In their digestion the hydrochloric acid is better combined, as a rule, than with the test breakfast. They contain also more gluten, which is utilized, when thus separated by the cooking with water, as well as the albumin of meat. The starch is also more freely liberated. Consequently, a greater percentage of nitrogenous matter is absorbed than with bread. The wheaten grits ferment more readily than the crust of roll and dry toast.

Rice is another cereal which contains starch in a digestible form, when properly cooked by prolonged steaming. It contains very little fat, and less albumin than wheat, but does not differ materially in its digestibility and physiological action from wheaten grits. Its composition constitutes it the best of the whole-grain cereals for obtaining the proper quantity of starch, which is very digestible when the rice is thoroughly cooked and passed through a fine sieve. Its starch is nearly all utilized, but its albumin is less easily digestible than that of wheat or corn bread. "Flaked rice" is a very digestible and palatable preparation.

Oatmeal contains a large quantity of fat, and more albumin and less starch than rice. Its high percentage of vegetable and badly-utilized fat constitutes a serious objection. It remains longer in the stomach than does rice or wheaten grits; it produces greater excitation and is more likely to undergo fermentation.

Cornmeal contains less fat than oatmeal, and in the form of thoroughly cooked, "mush" or "flaked hominy" is better utilized than any of the other cereals. More than 90 per cent. of both its albumin and fat are absorbed. It remains a little longer in the stomach than the preparations of wheat and rice. Its action on the stomach differs little from that of rice, but its intestinal digestion and absorption are better.

The cereals are not suitable for furnishing the body with its albumin and fat. From 10 to 75 per cent. of the albumin of the cereals is recoverable in the stools, but only two to five per cent. of the albumin of meats, fish, and eggs is lost. An exception should be made of the gluten preparations, which are valuable when a digestible form of albumin which resists putrefaction is required. Animal albumin, except cheese, putrefies much more readily than vegetable albumin. But these cereals contain the best form of starch.

In excessive secretion accompanied by fermentation these foods are imperfectly digested and the physiological action of their fermentation products is injurious. They should also be excluded in gastro-intestinal fermentation. In simple myasthenia, and in all diseases where the motor function is preserved, or where there is no excessive fermentation, they may be properly prescribed. They are sometimes not well borne when the stomach is morbidly sensitive. Intestinal putrefaction is no contraindication to their employment; and they combine rapidly large quantities of HCl, and leave the stomach early and easily.

Potatoes.—Another food which is sometimes prescribed, but which is rarely useful in the treatment of the diseases of the stomach, is the Irish potato. Only when steamed or baked and mealy, or boiled and mashed and again baked, with a little milk and butter, should it be permitted, until late in convalescence.

The potato excites a very active gastric secretion, the hydrochloric acid being left free very early in the evolution of digestion. The free acidity at the end of forty-five minutes is nearly double that of the test-breakfast at one hour. The motor function is also aroused, and the evacuation of the stomach is more rapid than with the cereals. The starch is badly utilized by the intestines, fermentation is favored and often initiated, and the small quantity of nitrogenous matter easily putrefies, so that the stools are foul, acid, and fermenting. The potato is valueless as a remedy in any digestive trouble, although it is a nutritious food when digested. The starch of the potato (seven per cent. lost) is not so well utilized as that of the preparations of wheat (one per cent. lost), rice (one to five per cent. lost), and cornmeal (three per cent. lost). One-third of the albumin of the potato, unless destroyed by putrefaction, is recoverable in the stools.

Fats.—The fats are not digested by the stomach, but they exert a decided action on this organ. The diseases of the stomach, except gastric retention, have no influence on the utilization of the fats, which is the work of the bile, pancreatic juice, and intestines. In large quantities, fat irritates the stomach, delays evacuation, and interferes with the digestion and utilization of albumin and the carbohydrates. The irritation, according to Leven, may be so great as to produce intense congestion of the mucous membrane. In small quantity, the best fats are without notable action on the healthy stomach when taken with other foods.

The action on the intestines varies with the kind and quantity of fat employed. By far the best utilized fats are butter, bone-marrow, cod-liver oil, pure olive oil, and cream. No other kinds of fat should be permitted in the diseases of the stomach. The products formed by germs out of fat are extremely irritating, and may produce auto-intoxication and enteritis.

As a physiological remedy, fat has no place in the treatment of the diseases of the stomach. The quantity should always be moderate, and in excessive secretion, in fermentation, and in many cases of myasthenia, it should be reduced to a minimum and the effect controlled by the clinical guides.

Sweets.—Like the fats, the sweets can not often be utilized as physiological remedies. Their action as such on the stomach and intestines is one of excitation of secretion and peristalsis. The action on the stomach varies according to the quantity and concentration. Large quantities produce a hydragogue effect, the secretion being poor in hydrochloric acid and ferments. In disease, their digestibility is greatly modified, and their easy fermentation constitutes the most serious objection to their employment. Concentrated solutions of sugar congeal or may even inflame the mucosa.

In the diseases of the stomach with excessive secretion, all sweets must be prohibited, as they only do harm, and are eventually lost as food where there is fermentation.

If the digestive tube is sweet, the motor function efficient, and the stomach is not morbidly sensitive or inflamed, sweets should be permitted. Their action in adenasthenia gastrica may be beneficial. Milk-sugar is a better remedy than the dextrinized cereals of commerce. It is very nutritious (teaspoonful or 10 gm. furnishes 41 Cal.), excites secretion but little when given well diluted and in small quantity, and is a valuable laxative food.

Green Vegetables.—Green vegetables, finely divided, tender, and thoroughly cooked, may be permitted in the diseases of the stomach where there is no excessive secretion nor retention. Given alone, they excite more gastric secretion than is required for their digestion, and remain a long time in the stomach. Their large indigestible residue and their resistance to fermentation make them valuable where constipation is troublesome. The best of these are spinach, tender string-beans, carrots, and lettuce as a salad. Asparagus and tomatoes, on account of their acids and the small seeds of the latter, may irritate a sensitive mucous membrane. The nutritive matter in the green vegetables is badly utilized.

Fruits.—Fruits, on account of their acid or sugar and their tendency to ferment, should be excluded in all diseases of the stomach where there is excessive secretion, morbid sensibility, and fermentation. As a rule, baked apples, ripe peaches, grapes, and prunes (which are among the most suitable) can be permitted in such diseases only when convalescence is well advanced.

The digestibility of food in health is modified by the diseases of the stomach. The digestive power of the stomach in each of its diseases will be discussed with the different diseases. It may here be once more emphatically stated that the digestibility of food in health is not the same as in

disease. It is only one guiding thread in the search for a proper diet.

3. *The physiological action of the food and of its digestive products should be such as to remedy or favor the disordered functions and anatomical lesions.* Food, in the treatment of the diseases of the stomach, should be used not only as a source of nourishment, but as a remedy exercising a special action on sensation, secretion, peristalsis, absorption, and the local circulation. The functional and bacteriological signs are guides in the selection of a digestible diet in the treatment of the diseases of the stomach; but the general digestibility of food in health is not so important as its digestibility in the particular diseases. Fortunately, the functional power in the well-defined diseases of the stomach is quite constant, and the diet should conform to this capability.

The functions of the stomach are germicidal, digestive, and absorptive. The germicidal activity seems to be dependent on the acid-secreting power, and whenever secretion is inactive special care should be taken to have the food and drinks sterile and sweet. No other dietetic indication is furnished by this protective function. Very little is known about the disorders of gastric absorption apart from its diminution in certain diseases. The acute diseases of the stomach are manifested by phenomena of irritation and demand a diet which gives protection and all possible functional rest. In the chronic diseases of the stomach the digestive function may be asthenic or hypersthenic. Asthenia may affect secretion or the motor activity and power of the stomach. Hypersthenia may affect sensation, secretion, and motor activity, and it is manifested by pain, nervous unrest, excessive or continuous secretion, and by spasm. The various functions may be separately affected or combinedly affected in the same manner, or one function may be asthenic and another may be hypersthenic. Whatever be the character of the abnormal variations, the diet should be appropriate to the disorders of sensation, of secretion, and of the motor function, and to the accompanying fermentation and putrefaction.

Morbid sensibility of the stomach is either a dynamic affection or a symptom of an anatomical disease. The hyperesthesia of organic disease is best treated by a bland, non-irritating diet. All alcoholic drinks should be excluded. Sour foods and acids are also pernicious, and no condiments except a small quantity of salt should be permitted. The temperature of the diet is also important, and the food and

drinks should be neither very hot nor cold. The fats, if perfectly fresh, are well borne, but the contrary is true of the sweets, particularly in large quantities. Meat broths and coffee should be excluded, and milk often disagrees. The meats should be finely divided and the young white meats rich in gelatin are to be preferred. To these should be added the finely-ground and thoroughly-cooked cereals and some of the green vegetables.

There is no fixed rule for the selection of a diet in morbid sensibility of the stomach (hyperesthesia and neurasthenia) as a dynamic affection. Here we have to deal with a neurosis extremely capricious in its likes and dislikes. Contrary to what seems reasonable, a soothing, indifferent diet often disagrees. This is particularly true of a milk-cure. Nothing but a trial can here guide us, and it is a good plan to begin with a non-irritating diet, and if this does not improve the symptoms, to substitute therefor, without hesitation, a diet physiologically more active and stimulating. Exclusive diets are rarely well borne in the nervous affections of the stomach, and an insufficiency of food is a grave error.

In excessive secretion, either of an organic or of a functional nature, the diet should be non-irritating, easily evacuated, and should possess a high acid-combining power. In adenohypersthenia gastrica all condiments and mechanically stimulating foods should be excluded. Adenohypersthenia gastrica may be converted by useless irritation into a gastritis. If the patient be anemic, exciting food is often sufficient to disturb the rhythm of the heart's action or to cause an attack of dyspnea; or, if weak, the overexcitation may produce tachycardia and induce an extreme state of depression. Under the circumstances, only harm can result from an excitant diet. On the contrary, the food should be non-irritating and as indifferent in its physiological action as it is possible to make it by minute division, solution, or suspension, by the removal of indigestible particles, and by the omission of condiments. The action of the food on the stomach should be reduced to a minimum in order to meet the physiological indication—which is sedative. The same rule applies to hypersthenic gastritis and to ulcer. In excessive secretion, of both organic and functional nature, the starches should be reduced to a minimum, and in severe cases be excluded, for the digestion of no other class of food leaves so much hydrochloric acid free. But functional and organic excessive secretion should not be treated in the same manner. In adenohypersthenia gastrica three moderately large meals should

be given daily, for a large proteid meal will leave less hydrochloric acid free and will produce less chemical irritation. Ulcer and hypersthenic gastritis, on the other hand, are properly treated when four or five small meals composed of bland, finely-divided food are ordered daily, for it is absolutely necessary to reduce mechanical as well as chemical irritation to a minimum. A milk-cure combined with alkalies may agree well, combining the acid rapidly and, without having produced irritation, leaving the stomach after a short sojourn; but milk, on account of the necessity of giving it at short intervals, allows the stomach little repose, and is often badly borne by the intestines. Consequently, a diet composed of a large quantity of finely-divided, lean, red meats, eggs, and a minimum of non-irritating cereals and fats may be more suitable. The sugars increase the quantity but not the acidity of the gastric juice. In small quantity and well diluted they should be permitted in organic and functional adenohypersthenia, provided the increase of secretion does not injuriously prolong digestion. Naturally, all irritants and stimulants should be excluded, and the diet made sufficient in quantity to support nutrition, aided, if need be, by absolute rest and rectal feeding. The two commanding indications are the control and utilization of the secretion and the protection of the sensitive mucous membrane against irritation. The object is not necessarily the diminution of the digestive work required of the stomach, but the regulation of the diet so that the stomach may painlessly perform such work as it is fitted to do.

In cases of simple subacidity, the greater part of albuminous digestion must be done by the intestines. The conditions are most favorable for the digestion of the starches. The digestion of the fats is but little interfered with. Consequently, the fats need not be diminished, and the decrease of the albumins should be made up for by a larger quantity of starches. The diet may be composed of meats, fish, cereals, vegetables, fruit, and digestible fats. No class of food need be excluded. Evidently, with the exception of the starches, nearly all the work of digestion is thrown on the intestines, and this necessitates very fine division and thorough cooking of the food. The diet should be digestible by the intestines, not prone to putrefaction like eggs, and should be so regulated in quantity and bulk as to preserve the motor function of the stomach. Milk is not well borne. The fats (butter, cream) should not be given in such large quantity as to produce diarrhea. Gastric stimulation, in

keeping with the cause of the subacidity, is advantageous, and consequently coffee, condiments, sweets, and alcoholic drinks in moderation may be beneficial.

The diet in the motor disorders is extremely important, and in this respect resembles the motor function itself. The chemical and absorptive work of the stomach can be performed by the intestines, and the compensation can be so complete as to maintain the organism in perfect health. The stomach must, however, do its own motor work, and the disorders of this function demonstrate in a striking manner the great harm which a diseased stomach can do.

In morbid muscular irritability of the stomach a bland diet is usually indicated, be the trouble vomiting or the too rapid evacuation of its contents into the intestines. But this is by no means always true, and a slightly stimulating diet may be best tolerated.

Myasthenia demands a very special diet—nutritious in small bulk, resisting fermentation, non-irritating, finely divided, soluble, sufficient in quantity, and exerting the proper physiological action. The quantity of water taken during the meal and during the period of gastric digestion should not exceed one glassful, for myasthenia is a "dyspepsia of liquids."

In myasthenia gastrica, if the patient is young and of strong constitution, and there is no contraindication, the excitant diet should be employed to awaken the muscular layer and to contract reflexly the abdominal muscles and increase abdominal tension. When the myasthenia is associated with excessive hydrochloric acidity, no starches should be permitted; and "peptones" and albumoses will be useful when hydrochloric acid secretion is diminished. If, on the other hand, the muscle is so exhausted as to be both weak and irritable, and if the muscular relaxation is due to inflammation of the mucous membrane, excitation would only cause tonic contraction or be ineffectual. Excitation would act on the stomach as does digitalis on a heart too weak to support it. Consequently, a bland diet is indicated in the treatment of the irritable and myasthenic stomach. It enables the nervous and neurasthenic to live without discomfort and without disordering reflexes. It relieves spasm and the stasis of contracture. The stomach churns and evacuates its contents, the bowels become regular, and the colon resumes its normal caliber. These effects are not theoretical, but can be demonstrated by clinical observation and abdominal palpation.

The disorders of absorption affect chiefly the utilization of alcohol and the absorbable sweets, which, when this function is in abeyance, remain too long in the stomach and produce irritation and undergo fermentation. Alcohol, however, stimulates this function in a state of health. Very little is known of the disorders of gastric absorption apart from its diminution in certain diseases, and, fortunately, the intestines are capable of doing all this work.

Gastric fermentation is a very common condition, and may require a very special diet or very rapid changes of exclusive diets. When this condition exists as a result of disease of the stomach and not of an improper diet alone, the treatment should be begun after very thorough lavage. Meats, gelatin, and dextrinized bread should form the basis of the diet. Green vegetables, fats, starchy foods, sweets, and fruits may be progressively added as the disease of which the fermentation is the result is improved by proper medication. Gastric putrefaction is very rare. It necessitates the careful selection, and sometimes the exclusion, of albuminous food. Fermentation and putrefaction are produced by certain germs in suitable soils. The dietetic treatment consists in making the contents of the stomach a bad medium for the growth of the particular germs which are found. It is a good rule of practice temporarily to exclude the food on which the germs are living. In rebellious cases we strongly recommend stomach washing and exclusive rectal feeding for a few days.

Gastric digestibility is the correlate of the digestive power of a particular stomach. The impairment of one or more of the functions in a particular manner affords so many guiding threads to the selection of a proper diet. In the dynamic affections it may be that the diet must be so regulated as to favor the stomach in every possible way, gentle or negative in its influence, or it may be that one or more of its functions need excitation. The indications for the use of diet as a remedy capable of soothing or exciting, are furnished by the subjective and functional signs. The stomach requires appropriate exercise and rest in order to regain lost power. Always to prescribe a favoring diet indifferent in its physiological action is a very serious mistake.

Not the dynamic affections only necessitate the selection of the diet with a view to its physiological action; the anatomical diseases of the stomach also demand either stimulation or rest. Chronic asthenic gastritis furnishes a good illustration of the value of an exciting diet, provided glandular degeneration is not so far advanced as to render stimulation

useless. On the other hand, acute gastritis imperatively demands temporary functional repose and the use of the blandest, most non-irritating foods. Ulcer and carcinoma should receive the most careful protection. In Sections IV and V the value and applications of the rule will be fully illustrated. The physiological action of the food and of its digestive products should be well considered in the selection of a diet, in order to avoid doing injury and to secure its full remedial influence.

The use of food as a remedy often consists in the ordering of a diet which exercises or even excites the functions of the stomach, or which excites one of the functions of the stomach and favors another. But not rarely gastric intolerance, or a gastric lesion, or gastric weakness makes it necessary to favor the stomach or to give it absolute functional rest.

The stomach may be favored by diminishing the nutritive needs of the organism, by throwing the burden of digestion on the intestines, and by rectal feeding.

By absolute rest in bed the nutritive needs of the organism may be diminished by about one-fifth, and the work required of the digestive organs is made just so much less. The diminution of the required digestive work may be necessitated by the inability of the digestive organs to utilize enough food to support nutrition, or by the desirability of protecting the stomach when it is the seat of a severe lesion, or by weak digestion, combined with great emaciation and debility.

The healthy intestines are capable of digesting enough food to maintain nutrition, and the stomach may, consequently, be favored by selecting an intestinal diet. The food should be very finely divided, liquid, concentrated, and bland, and it should be administered every two to four hours in small quantity. Milk, when it agrees, serves as an excellent basis for the diet. But by proper preparation many other kinds of food can be used. Expressed meat juice, meat powder, calf's foot jelly, meat jelly, cereals very finely ground and thoroughly cooked, are a few examples. Butter may be given with the cereals, or cream, and "vigor chocolate" and almond milk may furnish the requisite quantity of fat. The almond milk is prepared by making an emulsion of 20 almonds in a pint of hot water, and in proportion of one to two of milk is a palatable and nutritive liquid food. The prepared cereals and the albumoses of commerce are sometimes valuable. In this connection somatose and panopeptone merit special mention. But when peptones are prescribed, the total quantity of nitrogenous food for the twenty-four

hours must be reduced to a minimum in order to avoid producing diarrhea. Some of the peptones of commerce might be appropriately called purgative poisons.

Rectal feeding may be employed to favor and protect the stomach. Whenever it is not possible to nourish the body by the mouth, or whenever food administered by the mouth is liable to produce injury or to interfere with the cure of a disease of the stomach, complementary or exclusive rectal feeding should be tried.

The large bowel does very little digestive work, but it possesses the power of converting small quantities of starch and cane-sugar into grape-sugar. However, it absorbs water, sugar, dissolved albumin, albumoses, and emulsified fat. The absorption is not very rapid nor very great, and it is rarely possible to nourish a patient in even absolute repose for more than a few weeks by exclusive rectal feeding. Exceptionally, a patient with healthy digestive organs, and with a disease which kills only by starvation (as cicatricial obstruction of the esophagus), may be kept alive for about one year. In such cases it is probable that the enema is carried by antiperistalsis into the small bowel and there digested and absorbed.

Indeed, the investigations of Grützner make it probable that a part of a nutrient enema is absorbed by the small intestine. This antiperistalsis is favored by a weak salt solution (less than one per cent.), and explains satisfactorily the increased absorption and utilization of an enema which contains a pinch of salt.

Not enough nourishment can be introduced by the rectum to supply fully all the demands of nutrition; consequently, the body can be nourished by rectal feeding only imperfectly, and, usually, only for a short time. Enough water and salts can be absorbed by the colon, but the quantity of organic nutriment is altogether insufficient, except in very exceptional cases. Emulsionized fats, dissolved albumins, and their digestive products, alcohol, starch, and the absorbable sugars, may all find their way from the large bowel into the circulation, and be utilized in nutrition. The dissolved raw albumin, to which a little common salt is added, is as readily absorbed as albumoses and commercial peptones. Digested enemata possess no greater nutritive value than when undigested, and are prone to decomposition. They are not well tolerated, and often make the patients extremely restless and mentally and morally depressed. But however the nutrient enema be constituted, rectal feeding is likely to produce intolerance, and when too frequently repeated, and composed of pan-

creatized milk, may excite an acute colitis. Some patients resist this method of feeding, and are often unable to retain the enema. For these reasons rectal feeding is not popular, and is usually restricted in practice to cases of necessity. The method is employed much less frequently than it should be.

The valuable enema of Leube is prepared by mixing thoroughly—

150 gm. of beef pulp (no fat).
50 gm. of pulp of the fresh pancreas of the cow (no fat).
100 gm. of lukewarm water.

This mixture is warmed and injected slowly two or three times daily into the large bowel one hour after employing a cleansing enema of 250 gm. of lukewarm water. It does not irritate, the digestion taking place in the bowels. But the fresh pancreas is not easy to get, and the nourishment thus afforded is too exclusively albuminous. For 50 gm. of the beef pulp should be substituted the yellow of one egg, one tablespoonful of dextrinized (heat) flour, and one gm. of common table-salt. These should be stirred thoroughly into the lukewarm water.

The following preparation of Boas is well tolerated and is usually absorbed:

Milk,	250 gm.
Yolk of two eggs,	
Salt,	2 gm.
Claret,	Tablespoonful.
Prepared cereal food,	Tablespoonful.

Use one to three in twenty-four hours.

The preparation recommended by Ewald is also excellent. Two or three eggs are beaten up with a tablespoonful of cold water. About a tablespoonful of a prepared cereal (dextrinized) is boiled with one-half of a glass of a 20 per cent. solution of grape-sugar, and a wineglassful of claret is added. Let it stand until nearly lukewarm and slowly stir in the beaten eggs. Add one gm. of salt. Use two or three enemata a day.

These three excellent preparations may be employed in turn. If the contents of the large bowel become acid, both the milk and the sugar should be omitted, and as a substitute therefor beef tea may be used.

Practically, the greatest obstacle to rectal feeding is the rebellion of the bowel against the procedure. The rectal intolerance, by proper care and co-operation, may usually be

avoided. The enema should be warm, of the consistency of a thick soup, non-irritating, not larger than 250 gm., and given after careful cleansing of the bowel. One hour before the administration of the enema an injection of not more than 300 gm. of lukewarm water should be used; or, if the colon is already empty, the rectum should be simply washed out by the funnel-siphonage method. The nutrient enema is slowly introduced under low pressure, the tube is removed, and a soft towel is held for a short time gently but firmly pressed against the anus, while the patient remains quiet on the left side. A soft tube with a rounded end-opening should be used and introduced high up, or only above the sphincter if the effort to introduce the tube above the sigmoid flexure produces pain, resistance, and irritation. The enema introduced properly into the rectum will soon be carried by antiperistalsis, excited by the salt, high into the colon. Where the rectum is very irritable, a few drops of laudanum should be added to the enema. A nutrient enema should never be given oftener than three times daily.

In extreme conditions, a small quantity of nourishment may be given hypodermically. A few ounces of sterilized oil may be introduced in this way during the twenty-four hours, and, combined with whisky hypodermically, may aid rectal feeding in carrying the patient safely over a period of danger.

Rectal feeding may be employed to maintain nutrition and to give the stomach rest or protection. In cardiac and pyloric obstruction or stenosis it may be impossible completely to nourish the body by the mouth. Too little food reaches the intestines, and it may not be advisable to resort to surgery for relief. Whether the obstruction be malignant or benign in nature, life can be prolonged by the employment of nutrient enemata.

Rectal feeding is very valuable in the treatment of myasthenic retention. In this condition the body suffers both from lack of food and of water, and the slow starvation can be arrested by nutrient enemata, which compensate the gastric insufficiency. Rectal feeding is not only valuable on account of the support which it gives to nutrition, but also on account of the diminution of the functional work of the stomach. The favoring of the organ may be all that is required to relieve the retention and to give the stomach a short interval of repose. The complete evacuation also breaks the continuity of the germ growth. Even more effective is complete gastric rest for a few days. In myasthenic retention with active fermentation, if the patient be put to

bed, the stomach thoroughly washed out and daily douched internally, all food prohibited by the mouth, and the body nourished exclusively by nutrient enemata for from three to five days, the result is often remarkable. With this plan of treatment should be combined the daily use of the hot and cold needle-spray for one minute over the abdomen and lower extremities, external cathodal galvanization with large plate-electrodes, and faradization of the abdominal muscles. If the stomach does not retract, strychnin in full doses will prove, in some cases, a valuable aid. This short systematic medication, in which rectal feeding plays an essential part, rarely fails to control the fermentation and to relieve or diminish the retention. The same treatment is very valuable in myasthenic stagnation.

In the treatment of the dynamic affections where the stomach is morbidly sensitive and irritable, a few days of exclusive rectal feeding may be advisable. A good result is often thus obtainable in uncontrollable vomiting, in hyperesthesia, and in adenohypersthenia gastrica, the rectal feeding acting as a remedy by giving absolute rest to the stomach and furnishing some nourishment to the body.

In the treatment of obstinate and complicated ulcer of the stomach, rectal feeding may be used to supplement or to supplant feeding by the mouth. Hemorrhage and local peritonitis and perforation are stringent indications for exclusive rectal feeding. So great, indeed, are the advantages of a few days of gastric repose that even in simple, uncomplicated ulcer, in the beginning of the treatment, or intermittently during the cure, rectal feeding might be more frequently employed with benefit and without the least danger.

In severe acute gastritis and in the hypersthenic form of chronic gastritis, the incalculable value of gastric rest for a few days demands the employment of rectal feeding. Also in the diseases of the stomach which are not compensated by the intestines, and when too little food is digested and absorbed, rectal feeding aids in the maintenance of nutrition, particularly where the large bowel is healthy. Some good may also be done by this method of feeding in arresting the progressive inanition of carcinoma.

4. *Not only the diseases of the stomach, but the power and state of other organs, should be kept in mind.* The stomach decides the selection of the diet in only one condition—viz., when it is the only organ diseased. The dietist will here experience little difficulty in formulating his prescription in keeping with the indications given by the careful application of

the methods of investigation. Unfortunately, it is rare that the problem is so simple and easy of solution.

The intestines establish digestive compensation in the diseases of the stomach. Consequently, be the intestines healthy or diseased, they are never silent in the selection of a diet. The diet in a diseased stomach with normal intestines is, so far as the intestines are concerned, prophylactic. The food should be finely divided and free from abnormal chemical, physical, or living irritants. The meats should be palatable and freed from indigestible matter; the cereals should be finely pulverized and thoroughly cooked, and the fats sweet, and those most digestible should be selected. No food should be given that is likely to undergo fermentation in the stomach, or to throw extra work on the intestines, or to force them to work at a disadvantage. The intestines should be as closely watched and favored and protected as the heart-muscle in valvular lesions, for the intestines stand between the diseased stomach and inanition.

When the intestines, as well as the stomach, are diseased, the diet should be regulated in accordance with the digestive and assimilative power of the alimentary canal. The diet should favor the weakest point; what can not be digested need not be prescribed; the foods should be selected which have the best chance of escaping destructive changes, the physiological action of which on the diseased digestive tube is most favorable to their digestion and absorption, and the therapeutic effect of which is most likely to be beneficial. The task of the dietist is here a hard one, and can only be done by precise and correct directions, by close observation, and by free use of the clinical guides.

The diet in the diseases of the stomach should also be modified so as to be appropriate to any other organ that may be diseased. The diseases of nutrition, particularly gout and diabetes, should not be overlooked, and nephritis often forces a compromise. The waste products of the carbohydrates and fats no doubt give the kidneys less work to do than those of albumin, which latter may be retained in the blood in poisonous quantities. But the normal products of albuminous catabolism are much less injurious than the absorbed products of gastro-intestinal decomposition and some forms of fermentation. The first requisite of any diet is that it be digested and absorbed, and, consequently, the stomach and intestines, when diseased, always exercise a controlling influence in the selection of a proper diet. Other diseases may so modify a diet as to destroy its value as a physiological

remedy in the treatment of the diseases of the stomach. The remedial influence of food should then be replaced by other forms of appropriate medication.

5. *The finances, habits, and peculiarities of the patient should be considered.* In selecting a diet for the rich, with all the luxuries and comforts at their command, the foods which are to furnish nourishment and aid in the cure may be chosen from the best in the market. But many feel the sting of poverty, and must live on what is cheap. The practical physician will order the foods which are best and most suitable under the circumstances.

A man's stomach is often what he makes it. It readily becomes the slave of habit, and is often endowed by circumstances with peculiar capabilities and weaknesses. Foods agree and disagree contrary to what would rationally be expected. These results of training and these individual peculiarities should not be rudely disregarded, and the patient ordered to eat what agrees with most men afflicted with the same disease; a man is too prone to order others to eat what he himself best digests.

6. *The directions should be complete and explicit, and should be changed to meet the daily indications.* It is only when it is carefully and minutely regulated, and changed from day to day to meet the new conditions, that the value of the dietetic treatment of the diseases of the stomach becomes so striking. If the physician be content with writing a list of permitted and forbidden foods and drinks, and does not keep the patient under control and observation, no permanent benefit is likely to be conferred. It is necessary to watch the evolution of the disease, so as to be able and ready at the right moment to make the suitable changes in the diet.

In writing a prescription, it is usual to select the best drugs for the special case, to regulate the doses according to the effects desired, and to order them to be taken in a particular manner at regular intervals, until it is evident that the effects are obtained. The principles of prescribing a diet are the same. As a remedy, the diet should be used in the same manner as any article of the materia medica.

II. SIGNS OF CORRECTNESS OF THE PRESCRIBED DIET.

The diet being rationally selected in accordance with the rules already given, it should next be prescribed and controlled by the clinical guides.

If the diet is correct, the discomfort of which the patient complains will be relieved. There is something radically wrong with a diet which increases the subjective symptoms, and the patient will be the first under the circumstances to protest. When the diet does not diminish or relieve the discomfort of which the patient is conscious, all that can be said is that the diet is doing no harm. The correctness of the diet is proportionate to the relief afforded. It is not expected in every case that the trouble will be relieved as if by magic, but the sensations of the patient, where no indulgences are taken, form a rough but trustworthy clinical guide.

A correct diet will usually cause no loss of weight and strength; otherwise the first requisite of a diet—which is the maintenance of the nutrition of the body—is not fulfilled. But not rarely, even in the simple diseases of the stomach, the diet must be insufficient. The injury done to the organism must not be carried so far as to outweigh the benefit derived from the favoring and repose of the stomach. The diseases of the stomach with a tendency to emaciation, particularly the motor insufficiency of carcinoma, should never be treated by a reducing diet, for here the organism has no power to regain what is lost.

If the symptoms disappear without loss of weight, the diet is correct, and should not be changed unless it is necessary or advisable to force alimentation in order to improve the state of nutrition. If there is no loss of weight or strength, but no improvement of the symptoms, the diet is sufficient but not remedial. If the patient feels better, but weight is being lost, the diet, except in cases of obesity, is proper but insufficient. A diet which does not improve the symptoms, and also reduces the weight, should be considered as radically wrong. In retention and carcinoma it may not be possible to arrest the emaciation; the symptoms may, however, be improved by proper diet and medication. These simple rules are excellent clinical guides.

In addition to the sensations of the patient and the gain or loss of strength and weight, there are also other clinical guides to the correctness of the dietetic treatment. Among these are certain physical, functional, and bacteriological signs.

The abnormal physical signs, in so far as they are dependent on the diet, should improve, although they are often the expression of the lesion only. An increase or decrease of myasthenia can be recognized. Gastric flatulency, occurring immediately after a meal, when there is no gastric retention, is due to swallowed air, and if the stomach is much distended

is often a sign of myasthenia. Under these circumstances, if the diet is suitable to myasthenia, the flatulency is not a revealing sign of a dietetic error; but in the absence of gastric retention and colonic stagnation and fermentation, flatulent distention, occurring immediately after a meal, is a sign of myasthenia, and it should be at once considered whether the diet is not physically too heavy, or too large, or too exciting. Overstimulation of a myasthenic stomach produces irregular and often painful peristalsis or complete relaxation. The flabby wall yields readily to the expansibility of the contained gas.

Gastric flatulency, coming on some time after a meal, may be due to myasthenia, to gas-forming fermentation, or to putrefaction. Gastric putrefaction, except as an accident, is almost unknown, except in carcinoma and in gastric retention, and then it is always accompanied by fermentation. The gas in the stomach is either swallowed, regurgitated from the intestines, or formed in the stomach by fermentation. (The chemically-decomposed carbonates and exhaled gas from the blood may be disregarded.) The cause of the flatulent distention may be readily determined by using the stomach-tube or by giving an exclusively meat meal. If it be found due to fermentation, the diet must be so regulated as to avoid or control this abnormal process.

Flatulency is more often in the intestines, and when abnormally increased is a sign of increased gas formation by putrefaction and fermentation, or of this process combined with myasthenia, or of myasthenia alone. The stools and urine should be examined for signs of putrefaction; if negative, test the acidity of the stools and prescribe an exclusive meat diet as a trial. If either fermentation or putrefaction is found, the appropriate diet and medication should be prescribed. If neither exists in excess, the diet and medication are directed against the myasthenia.

The influence of the diet as a remedy is revealed by the functional signs. If the gastro-intestinal functions are improved thereby, the diet is correct. If the functional signs are made worse, and the other medication is not responsible, the diet is either wrong or, as regards the intestines, an enforced compromise. The intestines compensate a diseased stomach, and their functional activity requires careful preservation. A diet which does not maintain the functions of the intestines in their integrity is always regrettable, but is sometimes advisable in order to favor a diseased stomach temporarily. If there is no change in the functional signs, the diet

is either a failure or inactive as a remedy, but may be the best that is possible if it is in conformity with the other clinical guides.

The bacteriological signs are useful not only in diagnosis, but also in the selection and control of the diet. As a clinical dietetic guide, not only the bacteriology of the stomach, but also the evidences of excessive germ activity in the intestines, should be sought in the stools and urine. The diet should be so managed as to control the excessive germ growth in the digestive tube.

Naturally, the curative treatment is directed against the conditions which favor the excessive germ growth; but much can also be accomplished by a proper selection and by sudden changes in the diet so as to form an unfavorable culture soil. Fermentation and putrefaction reveal themselves in the odor, reaction, and composition of the stools. In the urine are found the products of the putrefaction, and of these products the one of most importance is indican.

The value of the quantity of the aromatic sulphates in the urine as a clinical guide is unquestionable. The quantity of indican—which may be taken as an indicator—is not always proportionate to the activity of intestinal putrefaction, but is also dependent on absorption. Only when absorption is good is it a guide. A negative result should not be given a positive meaning.

Intestinal putrefaction is most active in the ileum and colon and is greatly influenced by the acidity of the contents. This acidity is due to two factors—the secreted hydrochloric acid and the organic acids of fermentation.

To the dietist, the two most important circumstances which influence intestinal putrefaction are the acidity of the gastric secretion and the composition of the diet.

If intestinal and gastric fermentation or intestinal putrefaction be produced or increased by the diet, a mistake has been committed, and the proper correction should be made at once. Putrefaction is more injurious than fermentation, for it produces not only local trouble, but also serious systemic intoxication. Where these processes already exist, their decrease is the clinical sign of the correctness of the diet.

By the daily use of these clinical guides the diet, rationally selected, is controlled and made appropriate to the individual case. In order to obtain the full remedial influences of food no indulgences should be permitted, and other forms of appropriate medication should be employed at the same time.

CHAPTER III.

PHYSICAL REMEDIES.

THE physical remedies which are used most extensively in the general treatment of the diseases of the stomach are water, electricity, and massage. To these should be added the abdominal belts which are employed to give proper support to the stomach.

The Uses of Water.—In the treatment of the diseases of the stomach, water may be used internally—as a drink, or to wash out the stomach or to spray the mucous membrane of the stomach; or it may be employed externally.

Water is continuously eliminated by the kidneys, the skin, the lungs, and the secretion and excretions of the digestive system. This loss is constantly supplied by absorption. In health, elimination and absorption are so balanced as to maintain the percentage of water in the body at a constant standard. The excessive drinking of water only raises the percentage temporarily. Elimination rapidly reduces it to the normal percentage, which is about 63. This vital law renders it possible to wash out the system by the ingestion and absorption of large quantities of this solvent. The use of very large quantities of hot water is not indicated by disease of the stomach. Uricemia, gout, rheumatism, and auto-intoxication, may demand the eliminating action of water, and these troubles may be associated with gastric disease. Before ordering such treatment we must know the motor power of the stomach, which determines the manner of administration, and may prohibit the use of excessive quantities of water.

A diminution of the quantity of water causes the fat deposit to be utilized (Oertel), while an increase may have the opposite effect. In water insufficiency, as in all other forms of starvation, the body is forced to live on itself. The increased nitrogenous elimination after the ingestion of water in excess is temporary, and has been attributed solely to the more thorough removal of waste tissue products.

The action of plain drinking water on the stomach is dependent on the quantity and the temperature. Mineral waters are employed as physiological and chemical remedies, and are considered in the fifth and sixth chapters of this section of the book. Cold water is an intense excitant of

secretion when taken on an empty stomach, and the action of water within limits becomes less intense with the increase of the temperature. Cold water remains longer in the stomach than water at spring temperature, while hot water is evacuated very rapidly by the normal stomach. The greater the quantity of water taken, the longer it remains in the stomach; but large quantities may be drunk, without proportionately delaying evacuation, by drinking it in sips. The quantity of water absorbed from the stomach is exceedingly small. Hot water excites less gastric secretion, is more rapidly evacuated by the normal stomach, increases the flow of bile, is a better solvent, and, consequently, is the form of water most useful in the internal hydrotherapy of certain gastric troubles.

The soothing effects of hot water are sometimes useful in allaying the irritability of the stomach, and are a valuable means of arresting forms of vomiting. In excessive secretion, and in digestive superacidity associated with normal motor activity, a glass of hot water, sipped slowly an hour before meals, is a simple and excellent remedy.

In myasthenia it is necessary to avoid overloading the stomach, and the quantity of food and drinks required to maintain the balance of nutrition must be so introduced as to favor the weak muscle as much as possible. The action of the diet must be such as not to delay or make difficult its evacuation. In addition to selecting food, finely divided, rapidly evacuated, and nutritious in small bulk, the quantity of water taken with the meal must be limited. The remainder of water required to supply the needs of the organism may be so given as to secure a therapeutic effect. In myasthenia with normal or diminished secretion, a single glass, or less, of cold water, slowly sipped an hour before each meal, often exerts a tonic influence. In myasthenia associated with glandular irritation or delayed evolution of secretion, a single glass, or less, of hot water, an hour before each meal, has an undoubted beneficial action, but the hot water must be sipped slowly and the stomach be completely empty before the time for the meal. In myasthenia the total quantity of water allowed should be strictly limited to that required by the organism. The stomach must always be empty at the beginning of a meal and never overloaded with fluids. In gastric retention, due to whatever cause, a drink-cure of any kind whatsoever is absolutely contraindicated, and the administration of hot water before meals is obviously worse than useless. Neither should very hot drinks ever be

given in continuous supersecretion, in ulcer with a tendency to hemorrhage, or in cancer with hemorrhage or with retention.

The chief indication, however, for the drinking of hot water—natural or artificial—is gastritis. The use of hot water in gastritis should be more accurately limited, and should be confined to cases with the motor function still intact and capable of evacuating in one hour a glass of hot water slowly sipped on an empty stomach. It removes the excess of mucus and exerts a healing and soothing influence directly on the glandular layer. It is also claimed that it prevents excessive fermentation, but so long as there is no motor insufficiency there is no other organic disease of the stomach with so few germs as are found in gastritis. Where the fermentation is due to stagnation or retention, the drinking of hot water is contraindicated.

Stomach washing is a mechanical remedy that demands very special indications and has fallen somewhat into disrepute through its too frequent use in cases where it can serve no good purpose.

The employment of this remedy should be limited to three conditions: (1) Retention or stagnation of food and of digestive products; (2) retention of excessive secretions; (3) a rich and active germ growth.

1. There can be no question as to the value of the remedy when there is retention. A stomach which never completely empties itself never rests, and is placed in a most unfavorable condition for regaining its lost power. The retention may be due to obstruction or to a high degree of myasthenia.

In simple myasthenia with stagnation, lavage is worse than useless where there is no gastric fermentation. If employed before the stomach is empty it robs the organism of the nourishment which without interference would be delivered eventually to the intestines. If employed after the evacuation is complete, it is evident that no advantage is gained. In both cases it only stretches and irritates a weak muscle without improving the condition of the mucous membrane. The morning washing, too, is here useless, for the stomach is clean and empty and is retracted; the evening washing does no good and relieves no symptoms.

In myasthenia with stagnation of the severe type (the stomach emptying itself during the night, but not between meals), accompanied by fermentation simply, the stomach should be washed out before going to bed and left empty until

morning; but if accompanied by fermentation and excessive secretion, it is best to wash out the stomach before the evening meal.

In myasthenia with retention the circumstances are altered. The stomach never empties itself completely, and is too exhausted to retract. Germs find a rich and persistent culture soil. The nervous system is continuously excited and the secreting cells accumulate neither energy nor products. The most important indication is to give the organ rest by artificially emptying and cleaning it. In our opinion, the evening washing, as a rule, is decidedly best in myasthenic retention, on account of the rest which is thus secured for the organ and the patient and the long break made in the germ growth. The evening meal must then be light, such as is quickly digested and emptied by the normal stomach into the duodenum. Between four and five hours later the stomach may be washed out and left completely empty until morning; but if there be little fermentation it is better to do lavage in the morning and enable digestion and absorption to proceed during the night. The operation should be performed daily until the bacterial growth is checked and the fermentation controlled, never introducing more than a pint of water before allowing it to flow out, continuing the operation until the stomach is clean, making sure that the water does not accumulate by comparing the quantity withdrawn each time with that introduced, and leaving the stomach completely empty. After the first week lavage every second evening will often suffice.

In myasthenic retention accompanied by excessive secretion and fermentation, the stomach should be washed out twice daily—before the evening meal and in the morning before breakfast. This plan controls the fermentation and enables the system to utilize the greatest possible quantity of food. If the body, however, is well nourished, the stomach should be washed out before retiring and left empty so as to give the organ functional rest and protection from irritation during the night.

In obstructive retention there is uncompensating muscular hypertrophy. The muscle has become stronger and then failed. There is the same stringent indication for washing out the stomach as in myasthenic retention. The best time for performing the operation varies. If the obstruction be such that it can be overcome by increased muscular power,—as is frequently the case in gastric displacement, in traction constriction, and in relative muscular insufficiency,—the operation

should be performed in the evening. In obstructive retention due to an organic lesion the operation can only be palliative, and it may be wisest to secure as much nourishment as possible for the body by making the stomach work at night. The stomach should then be washed out in the morning; or if there be active fermentation or excessive secretion, lavage should be done both before the evening meal and before breakfast.

2. The retention of excessive secretion is a condition which may be rationally treated by lavage. The retained secretion may be the product of the general or specific activity of the glands of the stomach.

Specific secretion, normally intermittent, may become continuous. This by no means rare trouble is usually associated with myasthenia. The muscle seems often inactive rather than weak, and the condition is analogous to the motor insufficiency associated with excessive lactic acid formation in malignant disease. The glands of the organ get no rest and are continuously irritated by their own products. It is a good plan to wash out such a stomach with plain warm water until clean, in the morning before anything has been eaten, and then with a 1 : 1000 solution of nitrate of silver, the stomach being left empty for at least an hour after the operation.

The general mucous secretion may be excessive and may accumulate in the stomach in large quantity. A coating of mucus is normal and is a necessary protection, but when the mucus accumulates, be it swallowed or excessively secreted by the stomach, removal is necessary in order to avoid the chemical and physiological disturbance caused by it. The stomach should be washed clean and prepared to offer fresh secretions to the ingested food. It is well to wash out the stomach in the morning before breakfast with a warm alkaline solution.

3. Lavage may be employed to get rid of a very active germ growth even when there is no retention. Through survival of the fittest, a particular micro-organism, or a particular class of them, as revealed by the bacteriological signs, may become established in the stomach in virulent activity. Washing the stomach in the evening with large quantities of water (medicated, if desired) will aid in rendering it sweet. Lavage is most frequently required for this purpose in carcinoma and in acute indigestion associated with fermentation.

The technic of stomach washing is very simple. Two methods are in common use. A funnel, to which is attached a piece of rubber tubing about 2½ feet long, is connected by

a short glass tube with the previously introduced stomach-tube, or the whole instrument may be made in one piece. A pint of warm water is poured into the funnel, which is raised above the mouth, and the water gently flows in. Before the funnel is completely empty it is quickly lowered below the level of the stomach, and by siphonage the fluid is withdrawn. The quantity of fluid withdrawn is noted and compared with the quantity introduced, and the solid constituents should also receive our attention. The fluid is then emptied into the receptacle and the procedure repeated

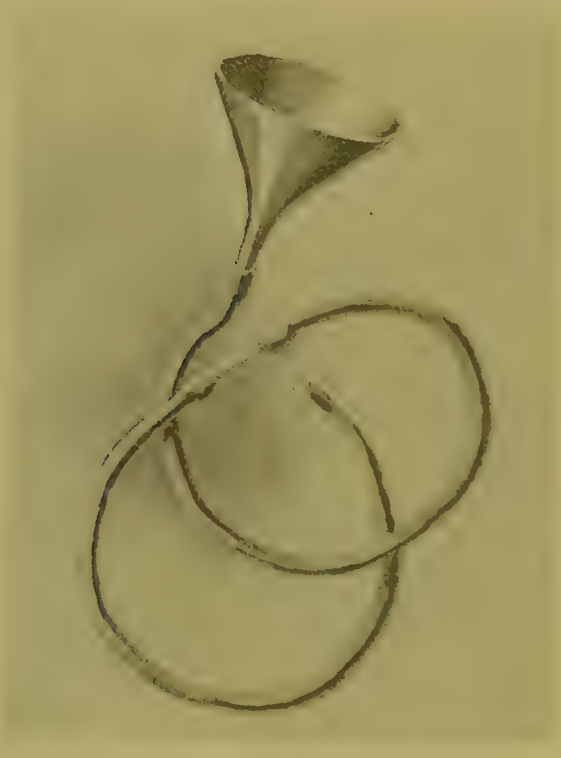


Fig. 13.—The simple apparatus of Somervail (1823).

until the viscus is clean. The last drop possible must be removed from the stomach by expression before the tube is withdrawn, so as to leave the organ empty. The introduction of a small quantity of water at a time, the avoidance of a strong inflow and of the accumulation of water in the stomach, and as complete evacuation as possible at the end are common rules which become imperative when there is myasthenia.

The second method is with a receptacle placed above the head. This receptacle is graduated and contains the water,

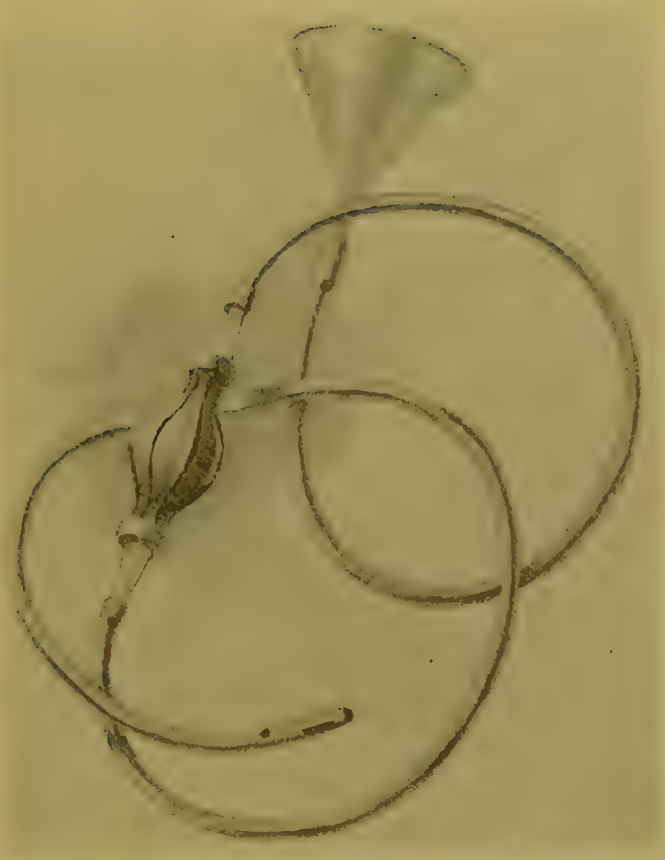


Fig. 14.—Friedlieb's apparatus for lavage and expression.



Fig. 15.—The Leube-Rosenthal apparatus for lavage.

and from the bottom runs a soft rubber tube to connect with one prong of a Y-shaped connecting glass piece. A second piece of rubber tubing is attached to the second corresponding prong, and ends in a graduated receptacle. On each of these tubes is fastened a clamp. The apparatus is connected with the introduced stomach-tube, and is then ready for use. The water is allowed to flow into the stomach in the desired quantity, while the outflow tube is closed. This is next

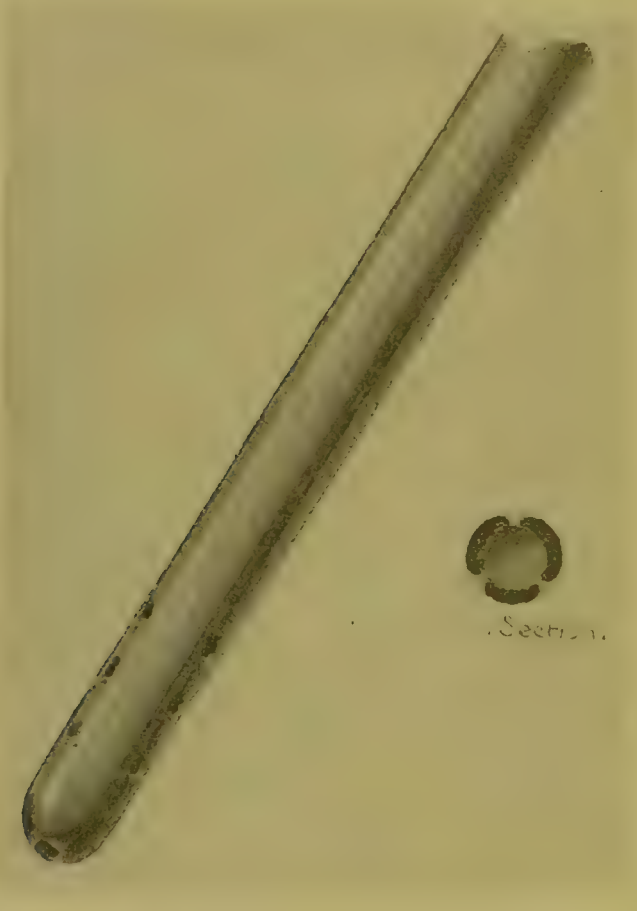


Fig. 16.—Rosenheim's intragastric douche tube.

opened and the feeding tube cut off. The contents of the stomach are aspirated automatically, and the procedure is repeated until the stomach is clean, when the tube may be withdrawn after the stomach is completely emptied by expression. If expression alone be inefficient, it must be aided by the position method.

The intragastric douche or spray is more beneficial in some of the diseases of the stomach than lavage. The spray

(Einhorn) possesses the advantage of distending the stomach so that the plain or medicated water can be brought into contact with all parts of the mucous membrane. The douche is the form which we prefer, but after the introduction of the tube the stomach should be moderately inflated with air before the water is allowed to flow in. The tube employed is the ordinary stomach-tube, which is, however, provided with a small end-opening and nine smaller openings on the sides. The edges of the openings should be rounded, and their combined caliber should be a little less than the caliber of the tube.

The douche is of service to arouse peristalsis (cold water), to increase secretion (teaspoonful of salt to a quart of water), to diminish secretion and allay irritability (nitrate of silver, 1 : 2000–5000), to excite the appetite (weak quassia infusion), and to disinfect the mucous membrane (permanganate of potash, 1 : 5000). The douche is employed when the stomach is clean and empty, and plain water is used before and after the introduction of the medicated solution, which should all be removed from the stomach.

Water may be used externally to exert an action on the nervous system, on nutrition, on the temperature of the body, and on the various functions. Indirectly, many of these procedures influence digestion favorably, but the description must be brief and limited to the uses of water in the treatment of the diseases of the stomach. This constitutes a special and important division of hydrotherapy.

The employment of any means as a remedy is based on its physiological action. Hydrotherapy, in addition to its general uses, may be employed to produce particular effects on particular organs. It is our object to describe how the action of water may be utilized in the treatment of the diseases of the stomach, be it our purpose to produce a particular effect on the nerve supply, on peristalsis, on the blood supply, or on secretion. In private practice the available methods are few and we seldom advise more than two—varieties of the compress and of the douche.

The compress may be used hot, cold, or of the temperature of the body.

The action of combined heat and moisture applied over the stomach is sedative—soothing the nerve supply, quieting peristalsis and spasm, and, when used for a long period, promoting the resolution of chronic inflammation. The hot compress is very beneficial in cardialgia, in spasm of the orifices and of the body of the stomach, in hyperesthesia

gastrica, and in similar conditions where sedation is indicated. The prolonged use of hot applications over the stomach is also beneficial in ulcer (no recent hemorrhage) and in chronic gastritis, but these applications do harm in acute gastritis and are dangerous in ulcer accompanied by hemorrhage. The digestion of very weak and emaciated patients is improved by moderately hot applications over the stomach during the digestive period.

The methods of employing combined heat and moisture are numerous. Towels wrung out of hot water and hot poultices are most commonly ordered; but the loss of heat and the necessity of frequent re-applications render these methods uncertain in their action and inconvenient. It is best to use an abdominal coil through which hot water flows constantly. Beneath the coil, over the abdomen, is placed a piece of flannel wrung out of hot water, evaporation being prevented by covering the whole with an impermeable tissue.

The cold-water application is antiphlogistic, and, when long continued, is also sedative. The most important uses of cold applications are to control acute inflammation (perigastritis) and to aid in the arrest of gastric hemorrhage. The cold compress is a most efficacious remedy in the treatment of digestive vomiting, and it is also beneficial in both acute and chronic gastritis.

The cold-water may be applied by means of wet towels or an ice-bag over a wet towel, or by means of ice-water running through an abdominal coil. To obtain the beneficial action of the cold compress it is necessary that the local action produce an active hyperemia of the skin. In the neurasthenic and anemic, and in very weak patients, the severe cold often produces passive congestion of the skin, and the action is injurious. To avoid this injury and action it is often necessary to place a rubber tube across the abdomen and beneath the coil, and through this tube hot water is kept constantly running (Winternitz). Whenever the disturbances against which the compress or local applications are directed are digestive, the applications should be made half an hour before meals, and should be continued during the period of digestion.

The Priessnitz compress, while applied cold or warm, is in reality a method of using water at the temperature of the body. It is sedative, soothing, antiphlogistic, and hypnotic in its influence, and it may be employed with a good prospect of benefit in all the painful diseases of the stomach.

The Priessnitz compress may be applied in several ways.

One end of a piece of flannel which is broad enough to cover the abdomen and long enough to go around the body and twice over the abdomen is wet in water of the desired temperature, and the bandage is applied and pinned in place. Or a towel may be wrung out of cold water, placed over the abdomen, covered with an impermeable tissue, and held in position by a flannel bandage encircling the body. The impermeable tissue prevents drying and lessens the chilling action of the cold. The compress may be worn day and night, and should be changed twice during the twenty-four hours.

In many of the diseases of the stomach the abdominal sympathetic is depressed and irritable. Neurasthenia gastrica is a disease which affects chiefly the nerves of the stomach. Many other diseases of the stomach, both organic and dynamic, can be favorably influenced by giving tone to the nerve supply, and hydrotherapy furnishes the most active remedy for the realization of this object. We refer to a special form of the needle-spray.

The intensity of the action of water on the nervous system is dependent on the temperature of the water, on the impressionability of the patient, on the mechanical excitation of the nerve endings, and on the location, extent, and duration of the application. The further the temperature of the water is above or below the indifferent point, the greater is the excitation. Consequently, a fine needle-spray momentarily applied under moderately high pressure, consisting alternately of hot and cold water, and extended over the area of distribution of all the nerves connected with the centers controlling the stomach, exerts a most powerful action on this organ. The needle-spray, applied in the manner which is here recommended, increases the activity of blood circulation in the spinal centers and in the mucous membrane of the stomach, and thus relieves their congestion and improves their nutrition, excites peristalsis and causes the stomach to contract firmly on its contents, and restores normal secretion and tones the abdominal sympathetic, as does no other single remedy.

The needle-spray should be used in the following manner, varying the temperature of the water, the hydrostatic pressure, and the duration of the bath so as to be appropriate to the individual case. The chest should be protected during the application, which should not extend above the level of the lower end of the sternum. The temperature of the water may be as high as 95° F. and as low as 60° F. The hot water is first used, and then, rapidly changing without gradua-

tion, the cold water is employed. The application is made with the hand nozle, revolving the spray for ten to twenty seconds over the abdomen, and then for the same time over the front and then the back of the lower extremities. The hot and the cold spray require only one-half to one minute each for their application. The douching is followed by rapid drying, brisk rubbing, and immediate dressing. The stomach should be empty when the bath is taken.

The Uses of Electricity.—Electricity is one of the most useful of the physical remedies in diseases of the stomach.

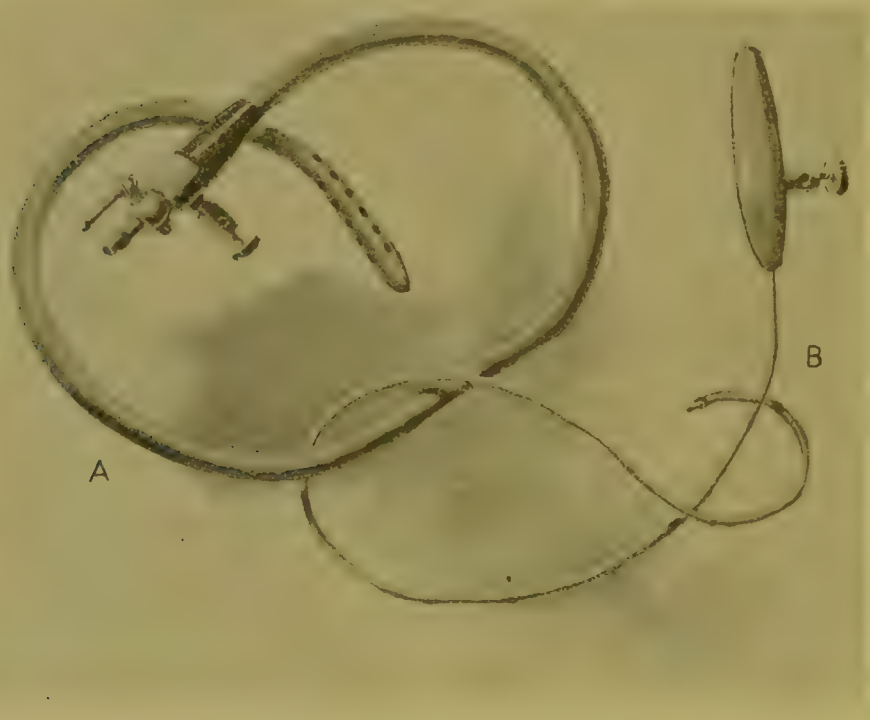


Fig. 17.—A, Rosenheim's intragastric electrode; B, Wegele's spiral electrode covered with rubber tubing, to be used in the ordinary stomach-tube.

Each physician has his own favorite methods of employing it. Only those will be described which in our own hands have given the best results.

Electricity may be applied internally or externally. The internal use is advisable in one disease and one condition—myasthenia—when the patient is accustomed to the use of the tube. Here the internal application would seem to be more powerful than the external; how much of the additional benefit is due to the more vivid impressions and suggestions it is very hard to estimate. The intragastric electrode of Rosenheim is

a very good instrument. Einhorn has invented a capsule electrode which is to be swallowed. The best intragastric electrode is the one recommended by Wegele. It consists of a stomach-tube through which runs a removable spiral conductor. The instrument can be kept clean and can be used to wash out the stomach, to introduce the water which serves as the intragastric electrode, and to remove the water after the sitting. The external method consists in the use of one or more forms of electricity in a particular manner—general faradization, central galvanization, and the local use of the two currents. Each method has its effects and its indications.

The effect of the galvanic and faradic currents on the secretions of the stomach may be considered well established by

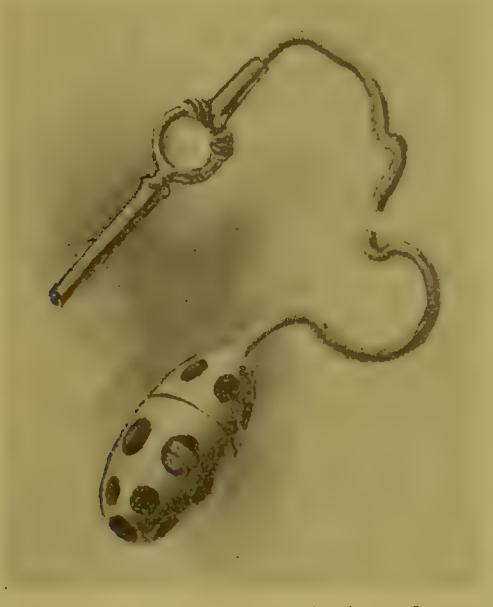


Fig. 18.—Einhorn's capsule electrode.

experiments, first on the dog and then on man, and by the analysis of the gastric secretion thus excited and obtained by the aid of the stomach-tube. After the use of a moderately strong current for about ten minutes, 20 or 30 c.c. of gastric juice may be obtained, rich in all the elements of specific secretion. Secretion may be excited both by external galvanization and by internal faradization. The motor function may also be notably excited—the contractions being tonic or peristaltic according to the strength of the current and the mode of application. Some deny that electricity excites contractions of the stomach. Meltzer laid bare the stomach of an animal and applied the electrode directly to its

wall. Faradization produced tonic contraction only when the electrode was near the pylorus; over the fundus the result was negative. With one electrode over the stomach and with the other either over the spine or within the stomach, no contraction of the stomach could be excited; but these experiments were made on anesthetized animals during shock. In spite of the denials of some clinicians, there is no doubt that electricity does produce tonic contraction and peristalsis of the human stomach. We have seen both galvanization and faradization cause gastric splashing to cease. When patients have thin abdominal walls, we have seen and felt the peristaltic contractions of the stomach produced by external galvanization, and these patients were not affected by peristaltic unrest (tormina ventriculi). A glass of water, the test-breakfast, and the test-dinner are evacuated earlier than normal by the electrified stomach. The effects of faradization and negative polar galvanization are greatest, both on secretion and on the movements of the stomach, after they have been employed for several successive days.

Electricity may also promote absorption, and is the one direct remedy which excites this function through its influence on the vasomotor nerves and the cells under the control and activity of which the process goes on.

But electricity is not only valuable on account of its action on the secretory, motor, and absorptive functions of the stomach. It can exert a favorable influence on the general sensations of the stomach, while it is very doubtful whether it has any direct action on the special sensations or the appetite.

This remedy also possesses an undoubted trophic action, which may be utilized in the treatment of the chronic anatomical diseases of the stomach.

Contraindications.—The contraindications to the employment of electricity in any form do not seem to have attracted much attention. The remedy may be harmful or useless.

1. It can only do harm in acute gastritis. It disturbs the organ when rest is imperatively demanded.

2. Electricity of high density does no perceptible good in severe degrees of myasthenia. It acts in the same manner as an excitant diet would under the circumstances.

3. The remedy should not be used during the active period of digestion. Theoretically, it would seem plausible that the functions of the stomach might be aided in the performance of digestive work; but practically the result of the combined action of the contents and of the remedy can not be con-

trolled. Electricity may, however, be given in myasthenia near the end of the digestive period.

4. A recent gastric hemorrhage is a contraindication. Neither should it be employed in diseases of the stomach commonly associated with hemorrhage. Chronic indolent ulcer may, however, form an exception where hemorrhage is not favored by the light hyperemia which accompanies the gentle electrization of the vagosympathetic with a view to obtaining a trophic influence.

We do not believe in the nihilism which claims that electricity acts solely through suggestion, or not at all. This may be true when the remedy is used without selection, method, or purpose. The physiological action of electricity and therapeutic experience should guide us in the choice and manipulation of the electrodes and in the determination of the form, strength, and density of the currents and of the duration and frequency of the sittings.

The best electrodes are the nickel-plated ones, with a half-inch thickness of sponge covered with linen. The electrodes are moistened in warm water. The supply should be large enough to enable us to use the requisite current strength and density and to apply them in the proper manner—continuous, interrupted, stable, or mobile.

The density of the current is represented by a fraction, the numerator of which is the current strength in milliamperes and the denominator, the surface area of the active electrode in square centimeters. The "active electrode" is the one over the point of greatest excitation, or over the central nervous system. If both the electrodes be over indifferent points, the mean surface area of the electrodes is taken. A current density of $\frac{1}{20}$ should not be exceeded in the electric treatment of the diseases of the stomach, and when the active pole is over the spine or neck it should not be greater than $\frac{1}{50}$. The selection of the poles in the employment of faradism is of no consequence; but galvanism possesses a decided polar difference, the negative pole being exciting and the positive pole sedative. To obtain the pure polar influence of the anode, only mild currents should be used, and care should be exercised in turning on and off the currents. Begin at zero and gradually and slowly increase to the desired strength; and at the end of the treatment the strength of the current must be decreased to zero in the same manner before the firmly-held electrodes are removed.

The duration of the sittings should be regulated according to the effect desired. Short applications are most generally

useful, and should never be so prolonged as to fatigue the excited part. Exhaustion is no remedy.

The frequency of the sittings is variable. In the painful affections the quieting influence of the anode may be required several times a day, and in the chronic diseases daily or weekly, and the electric treatment should be continued as long as it is beneficial.

Electricity may be used to produce: (1) Excitation; (2) sedation; and (3) a trophic influence. It may consequently be employed in the treatment of: (1) Asthenia gastrica; (2) hypersthenia gastrica; and (3) forms of gastritis and of ulcer.

1. The Electrical Treatment of Asthenia Gastrica.—Neurasthenia gastrica may or may not be associated with general neurasthenia, and, consequently, the treatment may be both local and general or local only.

The controlling indication is gentle excitation of the vago-sympathetic. For this purpose two local methods may be employed—the one indirect, through the connections of the sympathetic with the spinal nerves, and the other more direct, through the cervical sympathetic vagi connections and the gastric fibers running in the cord from the cranial cavity. For this purpose local galvanization or central galvanization may be employed.

A favorite method is a modification of the one recommended by von Ziemssen. Two large plate electrodes are employed with a surface area of 300 sq. cm. each. One is placed over the epigastrium, and extends from the pylorus across the abdomen to the left, covering the triangular area of the stomach in contact with the abdominal wall, which is marked by the lower border of the left lobe of the liver, the left costal border, and the line of the greater curvature. This electrode is attached to the negative pole, the object being to obtain the polar exciting influence. The other electrode, attached to the positive pole, is placed over the fundus, and extends to the left, toward, but not over, the vertebral column. The edges of the electrodes should be about an inch apart. The current strength should never exceed 15 milliamperes, which gives an electric density of $\frac{1}{20}$. The electrodes should be well moistened and held in firm contact with the body. The current should be turned on rapidly, allowed to flow constantly for about ten minutes, and should be shut off suddenly. It is not advisable to begin with a stronger current than five milliamperes ($D = \frac{1}{60}$), nor to continue the strongest permissible current (15), when it is used, more than a few minutes.

A second local method, which may be alternated with the above, is with the roller attached to the negative pole. The 100 sq. cm. anode is placed to the left of the dorsal spine. The current strength should be just sufficient to excite painless contractions of the abdominal muscles, and is usually about three milliamperes. The roller is moved, in firm contact, about 25 times from the fundus to the pylorus, along the axis of the stomach, and back again without lifting it from the abdomen, and about 25 slow strokes are made from above downward over the area of the stomach.

The first method influences the large sympathetic ganglia and the second excites more strongly the intramural ganglia of the stomach. These methods should be employed once or twice a week. The object will be defeated by meddlesome activity. If there be general neurasthenia, cervicogastric galvanization may be used at the same sitting. An electrode of 100 sq. cm. is placed over the triangular contact area of the stomach and attached to the negative pole. The other electrode, of 50 sq. cm., and attached to the positive pole, is placed over and between the fifth and seventh cervical vertebræ. The moist electrodes are firmly held in this position and the current is very slowly turned on; it should never exceed one milliampere in strength. The positive electrode is now slowly moved to the side of the neck, while firm contact is maintained, the upper border passing just below the mastoid process, and the electrode momentarily arrested when it has reached the anterior border of the sternocleidomastoid. From this location it is moved downward to the clavicle and along its upper border back to the starting-point. The same circuit is then made on the opposite side, each consuming about one minute, which, with one minute over the spine, makes the sitting last about three minutes. The current should be slowly turned off, unless spinogalvanization is to be employed. In this case, the anode is moved slowly downward to the end of the spinal cord and back again, consuming about three minutes, and the current is slowly turned off. In this method the density of the current should never be greater than $\frac{1}{50}$, the electrodes should be held in firm contact with the body, the duration of the sitting short, the applications should be separated by several days' intervals, and great care should be used to turn the current on and off slowly.

In the treatment of neurasthenia gastrica, a sitting may be given daily, and the sittings should be diminished in frequency as improvement takes place. In the electric treatment of the stomach and its connected ganglia, we

are decidedly in favor of small doses given in appropriate ways.

In myasthenia gastrica electricity may be used internally or externally. Internally, either faradism or galvanism may be employed. The stomach must be clean and resting, and about one pint of water is introduced or swallowed. A large plate electrode (200 sq. cm.) is placed over the epigastrium and the gastric tube electrode is introduced and connected with the negative pole and a current of from five to ten milliamperes is turned on. The external electrode may be moved during the sitting to the left and over the dorsal spine. The current is slowly turned off. Faradism, however, is preferable to galvanism, the external electrode being smaller (100 sq. cm.) and the current strong enough to produce painless muscular contractions. The external electrode may be held still, moved or stroked over the stomach, or held over the beginning of the dorsal spine. Intragastric electrization is rarely practicable. It can only be used after the patient has become accustomed to the tube, and this rarely happens except in conditions demanding frequent lavage, where electric treatment is often useless. The possible additional benefit derived from it is outweighed by the punishment inflicted; but the fatal objection is that the method is uncontrollable and the density of the current can not be regulated.

Another method of internal electrization of the digestive tube is more practicable, and may be often used in myasthenia gastro-intestinalis with excellent results. This is recto-abdominal galvanization. The rectal electrode, consisting of a short, rather stiff tube, is so arranged that the mucous membrane is protected against direct contact, and the water can be introduced through it. A very good electrode of this sort is that of Rosenheim. The tube is introduced and half a pint of warm water is allowed to flow in and to fill the rectum, in which the electrode terminates. To this is attached the positive pole. A plate electrode of 100 sq. cm. is placed over the epigastrium and connected with the negative pole. The current is turned on, and should not exceed five milliamperes ($D = \frac{1}{20}$); it is allowed to flow continuously for a short period and then should be interrupted a number of times. The external electrode is allowed to rest over the solar plexus and on either side of the umbilicus for about one minute. The whole sitting lasts about five minutes. A current strength of three milliamperes is generally sufficient. An application may be made every four or five days. Before the treatment the colon should be thoroughly emptied by a

saline or by an injection, and the rectum should be washed out before the introduction of the electrode.

A second method of treating myasthenia gastrica is with the faradic current. This current is very valuable when the abdominal wall is flabby. The proper external application of faradism excites secretion, peristalsis, and absorption and exercises the abdominal muscles. The current should be strong enough to produce painless contraction. One electrode of 100 sq. cm. is held to the left of the dorsal spine and another of 50 sq. cm. is applied over the epigastrium and the bellies of the abdominal muscles, with frequent interruptions. The sitting should last about ten minutes and may be repeated daily.

Adenasthenia gastrica without an anatomical lesion is very rare, but it may be met with as a result of shock or of hysteria. Spinogastric galvanization with the negative pole over the stomach may be beneficial, and should be combined with general galvanization of the spine or with general faradization. In the application of galvanism to the spine, the positive electrode of 50 sq. cm. is placed over the back of the neck and the negative electrode is moved from the upper dorsal region to the lower end of the cord. Acurrent strength of two milliamperes; slow increase from and decrease of the current to zero in the beginning and end of the sitting, which should last about two minutes and may be repeated daily, should be recommended.

2. Electricity in Hypersthenia Gastrica.—In hypersthenia gastrica the controlling indication is sedation, and we wish to obtain the quieting influence of electricity. The galvanic is the proper current, and should be so applied as to get the pure polar action of the anode. One plate electrode of 100 sq. cm. is placed over the upper part of the dorsal spine, and is connected with the negative pole. The other, of the same size, is held firmly over the stomach. The current strength shulod not exceed two milliamperes, and the current must be very slowly turned on and off. The sittings should be given daily, and should last five or ten minutes. This method of using electricity often gives most excellent results, and is employed in gastrospasm, in spasm of the orifices, in gastralgia, in habitual vomiting, and in excessive secretion, when not excited by irritant contents or by inflammation.

3. Electricity in Chronic Gastritis.—In chronic gastritis there may be either a state of irritation or of depression, according to the form and stage of the process. When the irritation is the expression of the lesion and not the result of irritant gas-

tric products, the sedative pure polar influence of the galvanic anode is very valuable. It is applied in the manner recommended in the treatment of hypersthenia gastrica, of which this is the inflammatory form.

If depression of function exist and the process, as indicated by the functional signs, is not so far advanced as to make excitation useless, either galvanism with both large plate electrodes over the stomach or spinogastric galvanization with the negative pole over the epigastrium should be used. The two methods are described under the electric treatment of neurasthenia gastrica.

The faradic brush mildly applied on the dry skin increases cutaneous sensibility and acts on deeper parts as a revulsive. It may be used to modify the circulation of the stomach. A plate electrode is held over an indifferent part, and the brush, with a current sufficiently strong to excite hyperemia but not much pain, is applied for a few minutes over the surface between the left sixth and tenth ribs, the median line, and the spinal column.

Electricity is a valuable remedy in the treatment of the diseases of the stomach when employed in a definite manner for a definite purpose. Its use, regardless of the form, strength, density, polar effect, points, and duration of the application, and in the fond hope of thus accomplishing something, is a waste of time.

Massage.—The physiological action of exercise differs in important particulars from that of massage, the first being voluntary, and the component parts of the body furnishing the source of power, while in massage the patient receives passively and is under the influence of force which has its origin in the well-directed efforts of another person. The voluntary muscular system furnishes about 45 per cent. of the total weight of the body, and while in action draws to it the arterial and drives out the venous blood. General exercise, built up into a system in the gymnasium, is a powerful revulsive, and draws away the blood from the internal congested organs; but it uses stored force and fills the blood with waste products. The weak may not be able to stand it, and it puts additional work on the eliminating organs. It excites chiefly the cerebrospinal nerves.

Abdominal massage uses little of the patient's force, forms little waste matter, and excites chiefly the abdominal sympathetic. It forces the lymph and the venous blood from the abdominal cavity and raises arterial pressure by filling the

arteries with the pressed-out fluids. It consequently purifies the tissues subjected to it.

Abdominal massage is decidedly an excitant remedy, and is contraindicated in all forms of gastric hypersthenia, of acute inflammation, of ulcer, of morbid sensibility, and of chronic inflammation associated with functional excitation. Massage can only cause cancer to grow and to spread more rapidly. It should never be performed while the stomach contains fermenting food, for it can do no good and disorders the intestines.

A great deal of harm can easily be done by routine abdominal massage, and the physician must prescribe the form and employ persons with special training to give it. The remedy may be used: (1) To empty the stomach; (2) to relieve an associated myasthenia intestinalis; (3) to strengthen the abdominal wall; and (4) to improve the abdominal circulation.

1. To empty the stomach by massage, procedures may be used which act reflexly or mechanically. The reflex-acting method utilizes the connection of the cutaneous nerves with the abdominal sympathetic, the gentle excitation of the skin being transmitted to the muscular layer of the digestive tube. The tip of the thumb rests lightly on the abdomen, between the symphysis and the navel, and the ends of the fingers rapidly circle about the umbilicus. No pressure is exercised, the tips of the fingers being brought in mere contact with the skin. A second circle may be made, more directly and exclusively over the stomach, with its central point over the left costal border half way between the ensiform process and the cartilage of the ninth left rib. Twelve or fifteen circles are made about each central point, and repeated after an interval of five minutes. The procedure is very useful to empty the stomach in myasthenia without retention. The patient soon learns how to do it intelligently. It should be employed after each meal, at the time when the stomach should normally be empty, the patient lying on the back with all clothing loose.

Reflex excitation of peristalsis may also be employed to strengthen the muscular layer in myasthenic retention. The stomach should previously be emptied by lavage, and a glass of cold water should be administered.

Zabludowski has recently described a good method of emptying the stomach mechanically. The mechanical method is used in the treatment of myasthenia, gastroptosis, and obstructive retention where the gastric muscle is insufficient

and quiet. If the muscular layer is hypertrophied, tonic, and powerful, massage can do no good. This muscular condition exists where there is obstructive retention, and is indicated by the increased resistance, by the higher percussion note, by the peristaltic sounds, and by the absence of splashing. The massage is intended to restore muscular compensation and to stretch the stricture of the pylorus due to non-malignant disease, the compressed contents acting as the instrument of dilatation. Manifestly, the massage expression through the pylorus should be employed only in special conditions. The stomach should be thoroughly washed out before the evening meal, which should consist of food in a state of fine subdivision. Four hours later massage expression may be employed and the stomach left retracted under the influence of circular cutaneous excitation.

The method recommended by Zabludowski follows the direction of the natural peristalsis from the fundus to the pylorus, and the manipulations are, briefly, as follows:

(*a*) A large fold of the abdominal wall and stomach is grasped between the thumb and fingers of the right hand as far to the left of the median line and as deep as possible; the contents thus caught up are thrown against the pylorus; this manipulation can be done successfully only when the abdominal wall is thin and flabby. The method of Cseri is better: The ulnar border of the left hand is pressed deep into the abdomen along the greater curvature of the stomach as it runs upward toward the pylorus; the pyloric end of the stomach lies in the palm of the left hand; the extended fingers and thumb of the right hand sink deep into the body of the stomach and with a pushing stroke force the contents toward the pylorus.

(*b*) Where the abdominal wall is thin and flabby, the stomach may be divided into two cavities by the fingers pressed with a kneading movement deep against the vertebral column. The contents of the pyloric division are pressed through the pylorus by moving the fingers to the right and upward, dilating the orifice like a bougie.

(*c*) The finger-tips of both hands are pushed deep into the abdomen along the left costal border over the stomach and the oscillating hands knead the contents toward the pylorus, while the fingers are kept constantly in vibration. This procedure is a powerful excitant of peristalsis.

(*d*) The stomach is grasped like the uterus in the placenta-expression method of Crédé, and the contents are squeezed through the pylorus.

The procedures should be alternated. The massage should never be painful and should be performed only while the muscles are relaxed. The treatment should never continue longer than ten minutes, the patient lying half the time on the back, and the other five minutes on the right side.

2. Myasthenia intestinalis is often associated with myasthenia gastrica. The massage of the stomach should then always be preceded by the mechanical emptying of the colon. The methods described by Reibmayer accomplish this object well. If the contents of the colon are fluid, the massage may begin with the cecum ; but if, as is commonly the case, there are solid fecal collections, the massage should begin with the descending colon. The manipulations are the following :

The right hand is laid flat over the descending colon, with the fingers outstretched and strengthened by the finger-tips of the left hand over the metacarpo-phalangeal joints. The hand is moved downward and inward, avoiding pressure against the iliac bone, the fingers following the dip of the sigmoid flexure into the pelvis.

The right hand, completely extended, is next placed over the cecum, the fingers covering the umbilicus, and the ulnar border is pressed deeper than the radial. In this position, and under slight pressure of the balls of the little finger and thumb (the other fingers remaining passive), the hand is moved along the ascending colon to the right costal border, across the abdomen in the course of the transverse colon, along the left costal border, and over the remaining part of the colon. Care must be used to avoid causing pain by pressure against the bony prominences.

To empty the cecum, the masseur turns his left side to the patient and places the right hand, with distended fingers directed toward the symphysis pubis, over the right iliac fossa. The fingers are strengthened by the tips of the fingers of the left hand applied over the metacarpo-phalangeal joints. The movement, with gentle pressure, is upward over the cecum and the ascending colon until the fingers reach the right costal border, and then inward, gradually relaxing the pressure as the fingers pass near the umbilicus, to the starting-point.

The mechanical emptying is completed by repeating the massage of the transverse and descending colon, and peristalsis is excited by the reflex-acting cutaneous circles around the umbilicus. Later, as the case improves, the solar and splanchnic plexuses may be vibrated, and the contents of the abdomen rolled between the vibrating hands.

3. To strengthen the abdominal wall the massage is limited

to the abdominal muscles, and a systematic series of active, passive, and resisted Swedish movements, aided by faradization, is employed.

Abdominal Belts.—The object of mechanical treatment by an abdominal belt is to increase abdominal tension and to support or hold in replacement the dislocated abdominal organs. In this way the vagosympathetic irritation is relieved and the disturbed blood and lymph circulations are restored.

The abdominal viscera are hung to the posterior abdominal wall and to the diaphragm above the plane in which they lie when the body is erect. Consequently, pressure or constriction at a higher point in the abdominal cavity than their location maintains and increases the downward displacement. It is, therefore, absolutely essential that the constrictions above the umbilicus and over the hypochondria should be removed. The stomach must be able to make room for itself on its own level as it becomes more and more distended with food, and not be forced to make room for itself by pressing and distending downward. No mechanical treatment can be of any avail so long as this supra-umbilical constriction exists, and an effort to support the abdominal organs will only result in the production of excessive abdominal tension and in the compression of the stomach.

The bandage must exert a proper degree of pressure from below upward and backward and hold the organs in reposition. The support should be very firm at the symphysis and lose itself on the normal level of the transverse colon.

There are many varieties of abdominal belts or bandages. The pelvic belt of Glénard is simple and sustains the abdomen. The modification of Montuuis is more suitable for some cases. The abdominal corset of Landau is excellent for increasing abdominal tension and for supplying an artificial abdominal wall. The bandages of Teufel, of Boas, of Rosenheim, and of Bardenheuer embody useful principles and often do well ; but it is best to have the bandage made to measure so as to fit properly, and to choose the form which best meets the mechanical indications of the particular case.

It is useless to apply a bandage with the organs in uncorrected displacement. The patient should assume the knee-chest position and the displacements should be corrected. The bandage is then tightened in place. If the organs are very movable,—floating,—they must first and always be replaced by one who knows how to do it. To attempt support without replacement is like fitting a truss on an unreduced hernia.

The bandage of support should be fitted on in the morning while the stomach is empty, and may be removed at night, while in the recumbent position, if abdominal tension is not very low.

The belt constitutes an artificial abdominal wall, and should be more and more elastic toward the upper border. The natural abdominal wall should be strengthened by massage and electricity and by very gentle active movements. The abdominal belt is an essential part of the treatment of gastroptosis.

CHAPTER IV.

SYMPTOMATIC TREATMENT.

THE object of the symptomatic treatment is the relief of the manifestations of the diseases of the stomach which give the patient special concern or discomfort. The most important of these symptoms which require relief are loss of appetite, pain, vomiting, and flatulency.

Loss of appetite, where there is no anatomical disease of the stomach, is often relieved by orexin. The basic orexin in fine powder should be given in a gelatin capsule in a single dose of from three to five grs., preferably during luncheon, or, as Penzoldt recommends, with a cup of bouillon at 10 A.M. The drug should not be continued longer than a week, and should never be given when there is an anatomical disease of the stomach or when the kidneys are not healthy. All medication which improves digestion increases the appetite, particularly the remedies which excite secretion and arrest fermentation or putrefaction. The appetite is also better when nutritive exchange is active, and proper exercise in the open air is one of the best means of increasing it.

Gastric pain may often be best relieved by the removal of its proximate cause. Neutralization or combination of the excessive hydrochloric acid may give relief, or the removal from the stomach of its irritating contents may be required. The latter is the only proper method when the pain is due to retention and fermentation. Gastric pain is often neuralgic in character. Antipyrin or one of the coal-tar analgesics will then give relief; aconite and gelsemium and also arsenic sometimes act well in the obstinate cases, and the specific

action of quinin in malarial neuralgia should be remembered. The quinin is best given combined with small doses of morphin. In hyperesthesia of the mucous membrane, nitrate of silver is both a symptomatic and curative remedy, but should be given only when the stomach is empty. Carbolic acid is a good temporary substitute, and bromid of strontium before meals is sometimes beneficial. The pain of the anatomical diseases is relieved by nothing so rapidly and completely as by morphin administered hypodermically. Codein is less efficient, but possesses some advantages. The method of administration is not a matter of indifference. Morphin given hypodermically is eliminated by the mucous membrane of the stomach, and about one-half of the injected dose can be recovered in the water used in the performance of lavage during the first two hours after the injection. The long-continued use of opiates diminishes the secretion of both the hydrochloric acid and the ferments, and leaves the mucous membrane hyperesthetic. A full dose of an opiate, however administered, diminishes secretion, delays the digestive transformation of the food (particularly starch), and decreases peristaltic activity and the tonicity of the stomach wall. The functions of the stomach are less disturbed by codein than by any of the other anodyne derivatives of opium, and the phosphate of codein (by mouth or hypodermically) is the best preparation. Belladonna diminishes starch digestion and prolongs the digestive period, but it controls excessive gastric secretion. Coca is an excellent gastric sedative when the pain is due to hyperesthesia of the mucous membrane, but it is inferior in this respect to nitrate of silver. A good preparation of cannabis indica is sedative and does not disturb digestion. Menthol is unreliable and we have often obtained no analgesic action from resorcin. The following tablet is an excellent gastric analgesic, to which codein may be added when the pain is spasmodic or very severe:

℞. Ext. belladonnæ alc.,
Ext. cannabis indicæ (Squibbs), . . āā . . . gr. $\frac{1}{10}$
Ext. cocæ, ... gr. iij.

In the treatment of all forms of gastric pain except that produced by a local irritant in the stomach, the soothing effect of hot external applications should be utilized. The neuralgic cases are sometimes relieved by anodal sedative galvanization.

The large number of remedies recommended for **vomiting** is good evidence of their inefficiency. Revulsion externally,

ice internally, sips of champagne, sips of hot water, cocain, carbolic acid, menthol, oxalate of cerium, small doses of ipecac, and sometimes opiates hypodermically or by rectum—all have their advocates. It is better to modify the treatment according to the cause. When due to gastric irritation, it is well to give large drafts of tepid water to wash out the stomach, or, when possible, use lavage. After the stomach is thoroughly emptied, an opiate should be administered hypodermically or by rectum. The vomiting of food may sometimes be controlled by applying the compress of Winternitz during digestion, or by the introduction of the food through a tube, or by Merck's resublimed resorcin. The treatment of this troublesome symptom is considered in the special part of this book, and most of the remedies likely to prove of value are given in the chapter on Habitual Vomiting.

The treatment of **gastric flatulency** is dependent on its cause. The gas may be formed by fermentation, or by decomposition of carbonates in the stomach, or may be swallowed with the food. Gaseous drinks and waters containing carbonates should be excluded. The amount of air swallowed with the food produces no disturbance unless the stomach is myasthenic. The treatment of flatulency thus reduces itself to the treatment of the fermentation or of the myasthenia. In either case, a glass of hot water will often enable the stomach to expel the gas. Abdominal massage will also cause the relaxed or distended stomach to contract and overcome the expansibility of the gas. Strychnin, however, is the sovereign remedy unless fermentation is the only cause. Stimulants and nervines of various sorts are advocated without good reason, and are administered with but little benefit. This chapter will be ended without apology, for we have a feeling akin to scorn against the symptomatic treatment of diseases of the stomach.

CHAPTER V.

PHYSIOLOGICAL TREATMENT.

Physiological treatment is based on the functional state of the diseased organ. It has to do with potency, with force; with an increase or decrease of vital power; with the abnormality displayed by the cell in the performance of its

special or its general work. Physiological treatment is directed against the quantitative or qualitative variation in the normal activity of the cell; against the excessive or the insufficient functional activity.

The abnormal functions, therefore, are either hypersthenic or asthenic, and the aim of medication is the correction of this or that state. A third possibility is the combination of the two; or, rather, lawless, variable, uncontrollable activity. The organ no longer works rhythmically. With the stomach this lawless activity is clinically a dynamic affection. In all cases the inconstant expression is due to the variable local excitation, and can not be considered a distinct pathological type. These variable conditions, consequently, do not demand special consideration. The physiological treatment is directed against the two states—hypersthenia and asthenia. The treatment, then, is either sedative or excitant.

Hypersthenia Gastrica.—The physiological treatment of this functional state is sedative, and is determined by the particular factor disordered,—sensation, secretion, motility,—by the genesis of the trouble, and by its dynamic or organic nature.

The indications may be thus enumerated: (1) Protection against irritation; (2) diminution of functional work; (3) sedative medication.

1. The irritants against which it is necessary to protect the hypersthenic stomach may be introduced with the food, or they may be formed in the stomach, or they may be prescribed as remedies. We have often emphasized the principle that the diet must be selected according to its physiological action. In hypersthenia the diet must be so compounded as to be as indifferent in its action as possible, and to combine the free hydrochloric acid.

Another source of gastric irritation is the accumulation of digestive products and secretions. Peptones excite free secretion, but the most excitant of digestive products are the sugars. Consequently, the accumulation of these products in myasthenia and in diminution of absorption should be controlled. Germ products are even more irritating than the digestive compounds. The protection of the stomach against irritation requires careful selection of the diet and of the drugs prescribed, prevention of stagnation and retention, and control of the germ growth.

On the same principle which requires the exclusion of irritants in the foods and drinks, and the prevention of stagnation and fermentation, all irritant drugs must be avoided.

The neglect of this precaution often leads to failure. Medicine is too frequently introduced into the stomach for some particular purpose, it may be, but in total disregard of its local action.

2. The diminution of the work of an excited organ is one of the fundamental principles of therapeutics. The irritable stomach deserves all the partiality that can be shown it. This indication may be met by an indifferent diet of mere support, including, in the severe cases, rectal feeding and absolute rest in bed.

3. The sedative medication may be internal or external. The uses of water and electricity have already been discussed, but a few drugs, also, are valuable gastric sedatives. Opium, coca, bismuth, aconite, veratrum viride, belong to this class. The prolonged use of the alkaline mineral waters in large doses is often beneficial, but the most reliable gastric sedative in the chronic diseases of the stomach is nitrate of silver.

The effect of the nitrate of silver is most manifest when the interior of the stomach is douched with a 1 : 2000 or 1 : 5000 solution. In the morning, when the stomach is empty, a pint of the solution is allowed to flow in through the stomach douche, and is then withdrawn after a momentary delay. To secure thorough removal the stomach may be afterward douched with plain warm water, and the residue after aspiration may be expressed. The application may be made once or twice a week until the morbid sensibility and excessive secretion subside. In supersecretion with stagnation, the douching should be preceded by lavage. The use of salt as a chemical antidote after the douching destroys the sedative action of the silver. The nitrate of silver may be administered by the mouth in solution, but is much less efficient and its local effect is accidental. Very little is likely to escape transformation into the inert chlorid or albuminate. Given in pill, it is rendered thus inert before it can come in contact with more than a very limited part of the mucous membrane. The stomach douche presents the most efficient and the only controllable method of using the drug as a local sedative.

The physiological treatment of hypersthenia gastrica is not the same in all its forms. When the excessive activity affects secretion or the muscular layer in a constant manner, it constitutes a particular dynamic affection or it is a sign of a particular anatomical disease of the stomach. The physiological treatment constitutes a part of the treatment of the different diseases and will be discussed in detail in the fourth and fifth sections.

Asthenia Gastrica.—The physiological treatment of asthenia gastrica has for its aim the restoration of the depressed functions; but not every asthenic state is to be treated in this way. The restoration must be proven possible before it is undertaken. Where disease has destroyed the noble elements such treatment would be useless. Where the depression is the result of active inflammation, excitation would only do harm.

A very valuable remedy in asthenia gastrica is the stomach douche. The douche should be given in the morning when the stomach is empty, the funnel being raised about three feet above the head of the patient so as to get a forcible rain douche. Plain water, physiological salt solution, or carbonic-acid water may be used. This is an excellent method of exciting the functions of the stomach in myasthenia, in adenasthenia, and in neurasthenia gastrica. The stomach should be left empty and quiet for half an hour afterward.

Food is one of the most powerful physiological remedies for the treatment of this condition. The diet is so selected as to exert by its action a stimulating effect on the motor, glandular, or nervous function. Instead of favoring the stomach, we push it on to its highest degree of capability.

The drugs employed in the physiological treatment of asthenia are commonly known as tonics. These preparations are often prescribed indiscriminately, with a view to building up the organism, but are indicated only in the curable states of depression, where a little excitation would do no harm.

The simple bitters have a very doubtful effect on the functions of the stomach. Given in large doses in health for a period of several weeks, they sometimes derange digestion. Their bad effect is rarely noticeable with small doses. The action on secretion is slight, possibly increasing it when it is normal or diminished. The motor function is uninfluenced or possibly stimulated. The excitant action of the simple bitters is more marked when they are combined with aromatics. The three most useful of the simple bitters are gentian, columbo, and condurango. Columbo acts more strongly on the movements of the intestines than do the other two, but all diminish the decomposition products in the urine. Condurango is also a sedative to the mucous membrane. In myasthenia with delayed evolution and excessive secretion, the fresh infusion, combined with nux vomica, is very valuable. Clinically, it is well established that the bitters sharpen the appetite and increase the secretion of saliva; and

it may often be observed that under their influence nutrition and the formed elements of the blood improve. The beneficial effects of the bitters are best obtained by administration a half hour before meals. It is probable that their effect is greater on the stomach after they are out of it, or the stomach is left by them in a state more susceptible to excitation. While in the stomach their action on secretion, according to well-conducted experiments, is no greater than that of distilled water.

Strychnin, on account of its more pronounced action, is of more value than the simple bitters. It increases muscular tone and excites secretion. Ipecac in very small doses has a similar action on the stomach, and the wine of ipecac may often be combined advantageously with the tincture of nux vomica; but when full doses of strychnin are desirable it is best to give the alkaloid alone. We frequently prescribe with excellent results in simple myasthenia and in adenasthenia gastrica a combination of these remedies in tablet form:

℞. Quininæ, ... gr. $\frac{1}{3}$
Ipecac., ... gr. $\frac{1}{10}$
Hydrastinin. muriat.,
Ext. nucis vomicæ, ... āā gr. $\frac{1}{20}$.

All hypersthenic states are contraindications to the employment of these drugs. Neither should they be prescribed in the anatomical diseases when excitation would do harm or be useless. A third contraindication is often overlooked. In neurasthenia gastrica they are badly borne, and in pronounced myasthenia they may convert a painless into a painful affection. Anything more than the mildest excitation is often injurious in myasthenia. In myasthenia of the colon a purgative and even a laxative dose of cascara will often leave an enterospasm which may continue for several hours. The descending and transverse colon, on palpation, may be then often felt as hard cords, which again dilate as the excessive irritation ceases. The myasthenic stomach may become contracted under the same influence as the intestine. Treatment which makes myasthenia painful is too stimulating, and the pain is the revealing sign of the injury being done.

Common salt and the bicarbonate of soda may be made to exert a very beneficial influence on the depression of the functions of the stomach. The bicarbonate of soda only acts as an excitant of secretion when given in very small doses before meals; five grs. in a wineglassful of water half an hour

before meals is sufficient. The salt should likewise be given in very small doses (three grs.), in about ½ per cent. solution, on an empty stomach. The small quantity of the weak solution acts after several days as a very energetic excitant. Large doses or strong solutions after short use will produce gastric catarrh. The sodium chlorid also stimulates peristalsis. A wineglassful of Saratoga Kissingen or Vichy sometimes give very satisfactory results, or other alkaline saline waters will do as well.

CHAPTER VI.

BACTERIOLOGICAL TREATMENT.

THE indications furnished by the bacteriological signs may be variously met by: (1) Chemical antisepsis; (2) removal and exclusion of the germs; (3) change of the culture soil and removal of the pathological conditions which favor germ growth.

Chemical antisepsis is with some practitioners a very popular method of treating gastric fermentation, but it is the least efficient and most injurious. The fermentation, not being an accident, but developing in consequence of the existence of favorable conditions, can be controlled only while the antifermentatives are present in the stomach. Antiseptics are also antipeptic, and irritate the mucous membrane, which in fermentation is already in a state of excitation. This method of treatment ordinarily deserves all the condemnation which can be heaped upon it, but at times some benefit is derived from the use of resublimed resorcin (Merck), of salicin (Merck), and of creosote.

The **removal of the germs** is more than a palliative measure, for it places the stomach in a position to perform its functions unmolested. For the safe and efficient removal of the germs there is no method comparable to stomach washing. As a remedy against excessive fermentation the operation should be thoroughly done according to the rules given in the chapter on Physical Remedies. Thus the continuity of germ development is rudely broken, and leaves comparatively few germs to struggle for existence on a much decreased amount of food. The exclusion of germs from the stomach

is an impossibility, but by most careful cleansing of the mouth and throat, and by selection of well-preserved and freshly-cooked food, much can be done to diminish the number which find entrance. The mouth should always be made sweet after the stomach washing.

A very valuable method of controlling fermentation is by sudden and complete **change of the culture soil.** The patient is placed for a day or two on an absolute nitrogenous diet, and then this diet is suddenly changed for one consisting of thoroughly cooked cereals, and again changed to a proper mixed diet. This method is more applicable in acute cases, where vomiting has partly emptied the stomach, a meat and egg diet being most suitable to begin with. In all cases the diet should be composed of such foods as are least liable to undergo the form of fermentation prevailing in the stomach.

Chemical antisepsis, lavage, and diet will prove only palliative if no change has been produced in the conditions which permit the excessive fermentation. The most common condition is motor insufficiency, and the promotion of the churning and evacuation of the stomach is one of the surest methods of keeping it clean. The fermentation will return as soon as the palliative remedies are stopped, provided the pathological conditions—such as motor insufficiency and excessive secretion—have not been discovered and successfully treated.

CHAPTER VII.

CHEMICAL TREATMENT.

THE chemical treatment has to do with the treatment of special symptoms by means of alkalies, acids, and the digestive ferments. The alkalies are employed to neutralize the excessive acidity of the contents of the stomach during digestion, while hydrochloric acid and the digestive ferments are very generally prescribed when the gastric juice is deficient. Consequently, chemical treatment may be required in hyperchylia and in hypochylia.

Hyperchylia.—In hyperchylia the chemical treatment is directed against the excessive hydrochloric acidity, which may irritate the gastric mucosa and may disorder intestinal

digestion. The excessive hydrochloric acidity is neutralized by the administration of alkalies at the right moment an din proper doses.

The alkalies have for a long time been employed empirically in the treatment of the diseases of the stomach, and their universal use would indicate the great esteem in which they are held by the profession. Their value in clinical medicine is due to their physiological action on secretion and on nutrition, more than to their properties as antacids. As antacids their effect is temporary and without permanent benefit, and unless they be given properly, they may be injurious.

The alkalies most in vogue are the sodium, magnesium, and calcium salts. The choice of the antacids is not arbitrary; for their neutralizing power and their action and the action of their chemical products on secretion and on the bowels may be important. Bicarbonate of soda combines less than half its own weight of HCl, and forms common salt and carbonic acid. Calcined magnesia combines nearly twice its weight of HCl, and forms magnesium chlorid. Ammonio-magnesium phosphate combines about its own weight of HCl, and forms magnesium and ammonium chlorids and phosphoric acid. The chlorids of soda and of magnesium are much less energetic in their action on secretion and are much less irritating than are the chlorids of calcium and of ammonium. From the ammonio-magnesium phosphate is liberated phosphoric acid, which is nearly as strong as HCl. As antacids in the treatment of hyperchylia, we consequently prefer magnesia usta and bicarbonate of soda; prescribing enough of the magnesia to regulate the bowels, and correcting its overaction by combining bismuth subnitrate with it. Only enough of the antacids should be given to neutralize the excess of HCl and to control the pain of hyperchylia. The small doses may be repeated during the free HCl stage of digestion, but it is rarely advisable to attempt to neutralize the gastric contents completely. The HCl which is combined with albumin should not be neutralized.

Hypochylia.—Hypochylia is very common, and it may be beneficial to supply the deficiency of secretion by the administration of hydrochloric acid and pepsin during digestion. This method of treatment is very popular. Physicians encourage it, it seems very rational to the laity, and druggists flood the market with digestive mixtures. We assert with emphasis that this supplementary chemical treatment has no curative value, is of restricted temporary utility in even suitable cases, and it may do positive harm.

Hydrochloric acid administered during the period of gastric digestion disappears very rapidly from the stomach, and it should always be given in combination with pepsin. Both pepsin and hydrochloric acid are necessary for the peptonization of albumin, and the acid and ferment are administered proportionately, or nearly so, in hypochylia.

The acid-pepsin combination should be given only when secretion is deficient and during the period of gastric digestion. The time of administration and the dose should be regulated by the functional signs and by the quantity and quality of the diet. The greater the quantity of albumin in the meal, the larger should be the doses and the sooner should their administration begin. If the meal consists largely of non-nitrogenous food, the smaller should be the doses and the later they should be given. The supply thus meets the physiological demand, and an opportunity is given for salivary digestion.

In our experience the best results are obtained by repeated doses during the period of gastric digestion. This period has no fixed length, but varies with the character of the meal. After a light meal that remains in the stomach about three hours two doses will usually suffice—half an hour and an hour after eating. After the chief meal, one or two more doses may be given, an hour apart. In our experience it is unnecessary to give enough pepsin and acid to completely digest all the albumin in the meal.

The supplementary chemical treatment may be employed when the disease of the mucosa permits a certain amount of excitation. When the mucous membrane should be given rest and protection, the administration of acid and pepsin and of predigested foods is injurious. The supplementary chemical treatment may be used when the work of the intestines, on the efficiency of which the maintenance of the balance of nutrition depends, must be lightened; but acid and pepsin, and the vegetable ferments which act independently of the presence of free hydrochloric acid, often disorder intestinal digestion and increase intestinal putrefaction.

SECTION IV.

THE DYNAMIC AFFECTIONS OF THE STOMACH.

All the disorders of the stomach which present no characteristic pathological anatomy are classified as dynamic affections. The displaced stomach may become diseased, or the diseased stomach may become displaced, and it matters not whether the disease of the stomach be anatomical or dynamic. It may be thought preferable, when a displacement of the stomach exists, to consider, for the sake of simplicity, the dynamic affection as a complication or as an accidental association. In like manner an anatomical disease of the stomach may coexist with a dynamic affection, and the anatomical disease may develop either before or during the course of the dynamic affection. The symptoms are then the expression of the dynamic affection combined with the symptoms and signs of the anatomical lesion. These complex cases should receive recognition, and they are not rare, for disease often defies simplicity and exclusiveness. The dynamic affections have no characteristic pathological anatomy, but their existence is neither destroyed nor excluded by the presence of anatomical lesions of the stomach. The anatomical lesions may coexist with the dynamic affection, but they constitute a separate and distinct disease. Some of the dynamic affections may be described as "neuroses of the stomach"; some of them are "functional disorders"; but the dynamic affections of the stomach may be in their nature neither the one nor the other.

Practically, this is one of the most important sections of the pathology of the stomach. Here disease presents itself in its genesis, and the subsequent evolution, unless arrested by proper treatment, may be represented by an inverted pyramid. The trouble grows along deviating lines, and becomes more and more irresistible. The physiological functions, one after the other, are enlisted with the forces which make for disease. The disease should be recognized and crushed in its pre-anatomical stage if permanent damage is to be avoided. This is the period when treatment will show its greatest power and give the most brilliant results.

A general characteristic of these affections is the specialization of the unhealthy variation. The disease dissociates the functions of the stomach and affects some one of them persistently and in a particular manner. Now it is a disorder of secretion or a motor trouble; now it is a painful increase of general sensibility or a particular sensation of the stomach which demands attention. Digestion may be chemically perfect or the stomach may churn and evacuate its contents unconsciously, the appetite being good. One function is persistently disordered; the others remain intact. The affection may be paroxysmal or intermittent, but the variation from the normal always recurs in a particular case in the same manner. In the course of the disease, however, the tendency is to spread to the other functions, and the primary dynamic affection may be supplanted or overshadowed by another. The true dynamic affections of the stomach never appear "like a panorama with ever-changing scenes." Such disorders are symptoms and nothing more. To consider the variable gastric expression of a disease of another organ as a distinct morbid entity is manifestly erroneous. The gastric disturbances cease when the exciting disease is cured. A true dynamic affection is capable of an independent existence, be it primary or be it secondary.

The manner of development is often peculiar. Suddenly beginning, without any dietetic error, and persisting for a variable length of time, the end may be no less sudden; or slow in its commencement, progressing by leaps, the termination comes when least expected. The unhealthy variation is not, or may not be, persistent, but intermittent. Rapid changes for better or for worse, without a perceptibly adequate cause, are characteristic. This obscure peculiarity is generally a contribution from the abdominal sympathetic, or from the brain, or from the general but temporary state of the body.

The dynamic affections may be in no close relation with the digestive act or with the alimentation. The symptoms may exist only or chiefly during the digestive period in some of the cases; but it is also true that the symptoms may occur when the stomach is empty. Digestion as a chemical process may be perfect, and the digestive disorder may exist only because the patient suffers and complains during the period of functional activity of the stomach. A large, a small, a mixed, and a simple meal may be equally well or badly digested. The physical state of the food may be of no greater influence than in health. There may be no constant relation between alimentation and digestion on the one hand and the

symptoms on the other. Nutrition may be well preserved, and sometimes even in spite of insufficient alimentation, although subnutrition (pain, vomiting, starvation) is sometimes as marked as in the grave anatomical diseases of the stomach. These peculiarities are often due to the existence of a trouble in some other part of the body which selects the stomach as the center of its manifestation.

The dynamic affections of the stomach develop preferably in a particular soil. This is the neurotic or nervous temperament or constitution. Some persons are born with a delicate and overwrought nervous system, and others acquire it by the mode of life, by bad habits, and by too exclusive and excessive mental or moral development. The resulting condition is unstable nervous equilibrium—the forerunner often of the nervous affections of the stomach.

Most of these affections are more frequent in women than in men, the proportion being about one to ten in youth, and gradually changing until the sexual difference becomes much less (about one to two) during the last third of life. The chief exciting causes in men are mental overwork and the reverses of fortune. The dynamic affections are common among women near the end of the social season in large cities, but are infrequent among country girls; for in spite of the pure air and sunshine of the country and the freedom from the withering touch of "culture," anxiety and sorrow and disappointment, and disorders of menstruation, of reproduction, and of lactation, have the same influence in the hut as in the palace. The predominance in women may be explained in part by their mode of life, their delicate organization, and their peculiar diseases.

The marked influence of the mind and feelings on the functions of the stomach is a fact long established by observation. Physiology and anatomy have given no satisfactory explanation of this intimate relation of the central and sympathetic systems; but the recent studies of Kölliker and others have thrown some light on this obscure matter. The sympathetic is a richly ringed chain of unipolar and multipolar cells connecting with each other and directly and indirectly with the central cells of the cerebrospinal system. The units of both systems have psychic and somatic functions. As regards the central nervous system, the sympathetic cells are of two kinds—the dependent and the independent. The independent ganglionic cells are chiefly motor, and innervate the whole involuntary muscular system, giving tonicity, producing contractions of the muscular fibers, and probably regulating also

the caliber of the blood-vessels. Some of these cells are probably sensory, and influence secretion. The dependent cells are in part sensory, and convey, particularly in disease, the indistinct visceral impressions to consciousness; and in part are motor, and enable the central system to influence indirectly, through the ganglia, both the involuntary muscles and the secreting glands. This discloses, in brief, the anatomical basis of the fact long known to the clinician—the influence of the mind and feelings on the functions of the stomach.

In many of the dynamic affections of the stomach the nervous system is disturbed out of all proportion to the local trouble, and insomnia, disorders of sensation, tachycardia, faintness, dilatation of the pupils, hot flushes, and blushing are very common. In health the organ does its work silently, but when the sympathetic is irritable, digestion produces an indefinable discomfort, and distant associated disorders develop. The symptoms are not characteristic, and they may be very similar in the anatomical diseases to the manifestations of the dynamic affections. In the one, however, is the constant and controlling and palpable anatomical lesion, and in the other is the invisible abnormality, recognizable only by its effects.

CHAPTER I.

THE SENSORY DYNAMIC AFFECTIONS.

I. BULIMIA.

The nervous affection of the stomach characterized by a sudden, imperative desire for food, and occurring in paroxysms and more frequently than in health, and greater than the needs of nutrition, is known as bulimia (βοῦς = ox, and λιμός = hunger). Other names for the same affection are hyperorexia (ὑπέρ = excessive, and ὄρεξις = appetite) and cynorexia (κύων = dog, and ὄρεξις = appetite).

Etiology.—Bulimia is most frequent between the ages of fifteen and forty, and in the female sex. It occurs as a symptom or association of some cases of ulcer, of functional adenohypersthenia, of hypersthenic gastritis, and, very rarely, of carcinoma and of obstructive and myasthenic retention. In mental diseases, and in organic cerebral troubles, a noteworthy percentage of the cases occur. Hysteria and neurasthenia are responsible for some of the cases. Disease of the male and female sexual organs may excite the affection. Syphilographers report some cases occurring during the third and sixth months of syphilis. Basedow's disease, intestinal worms, pulmonary tuberculosis (first and second stages), and pregnancy are found among the causes. Rupture of the thoracic duct, tuberculosis of the mesenteric glands, menorrhagia, intestinal fistula, and intestinal hypermotility may cause excessive hunger; but it is not evident that the desire for food is out of proportion to the needs of nutrition. Bulimia may be central, reflex, nutritive, symptomatic, and idiopathic. As an unassociated affection of the stomach, it is most common in the neuropath.

Pathology.—The pathology of the affection is obscure. Irritation of the encephalic hunger-center seems a plausible explanation of the cases due to diseases of the brain and of nutrition. Other cases appear to be due to the irritation of the nerve-endings or centers of the vagosympathetic. Hunger is normally excited by the action on the medullary hunger-center of the changed quality or quantity of the blood.

It would seem that bulimia is not excited in this manner, for it may be quelled by food in the stomach before there has been time for its absorption, and the attacks are in no relation to the composition of the blood or to the needs of nutrition; but it should be remembered that sensations in the stomach may inhibit the hunger-center or they may excite it. There is nothing against the supposition that either peripheral or central causes may be present and active. Clinically, there is no doubt that this pathological, excessive, imperative hunger is expressed by sensations located in the stomach, and it may be accompanied by the very rapid evacuation of the contents of the stomach into the duodenum.

Clinical Description.—In the mild form, a sudden, strong desire for food may occur at any moment during the day or night, while the stomach is full and active, or in repose. There is a little discomfort and uneasiness, possibly a little headache or vertigo, a slight burning or gnawing sensation in the stomach, but the desire for food is not imperative, although it is exaggerated, and the attack passes off after a little food or drink is taken into the stomach, and, after intervals of very variable length, may return suddenly, unexpectedly, and inexplicably.

The severe form is a much more serious affection. The beginning is sudden, inexplicable, and violent, occurring soon after a meal, while the stomach is empty, during the day or night. The affection may be continuous, the only relief being obtained during a short period after eating; this clinical form may be accompanied by violent exacerbations, which are most frequent in the forenoon.

The attacks may also be irregular or periodical, and the course intermittent, with short or with very long intervals. This lawlessness, or absolute disregard for all rules, is a distinctive characteristic. If the desire is not satisfied, the peculiar, indefinable visceral sensations become more and more unbearable, there are burning and pain in the stomach, and there may be headache, ringing in the ears, and vertigo; or the face may become pale, the extremities cold, and the patient may faint. The desire may become so strong as to overpower the moral sense, and food be taken regardless of its quality or ownership or of the surroundings. After the taking of food the sensations subside, but the effect is independent of the quantity, the quality, and the nutritive value of the food. The attacks may be quelled by eating a little food, or very large quantities may be required to still the hunger. But often the bulimia soon returns, imperative in

its strength, and without apparent reason. In the intervals between the attacks the appetite may be normal, or the severe attacks may be followed by loss of appetite, the hunger-center being apparently exhausted or completely inhibited.

The functions of the stomach may be normal or the evacuation of the contents may be too rapid. The affection is frequently associated with excessive hydrochloric acidity, but this functional adenohypersthenia may alternate with normal secretion. At least one important functional disturbing influence is irregular and immoderate eating. The course of the affection is variable, and it may terminate in spite of the persistence of the causative disease or condition. It is rebellious when due to encephalic disease, and it may disappear spontaneously in hysteria. Bulimia is sometimes the cause of myasthenia gastrica, of gastritis, and of intestinal disease.

Differential Diagnosis.—Acoria, polyphagia, and bulimia are often confounded. In acoria, the food eaten does not satisfy; the patient never feels that he has enough, while the desire for food may be normal or less than normal. In polyphagia there is a good appetite associated with a delayed feeling of satisfaction; or, at least, the sensation of having eaten enough does not become so strong as to interfere with the enjoyment of eating more—this is common gluttony. In bulimia there is strong, imperative, often unbearable hunger, which may be satisfied. From the form of gastralgia which occurs only when the stomach is empty it is distinguished by the strong and imperative appetite which accompanies the pain, and by the occurrence of some of the attacks when the stomach still contains food.

Treatment.—The etiological treatment is no less essential in bulimia than in the other nervous affections of the stomach. The hysteria or neurasthenia may demand a combined systematic cure, consisting of isolation, rest, diet, massage, electricity, hydrotherapy, and suggestive moral control. The other causative diseases require particular medication, a mere outline of which would here be out of place.

Antipyrin, or a similar analgesic, may moderate the severity of the attack. Opium or codein may also be used. Both of these drugs are valuable in the diabetic form. The bromids of strontium and arsenic are palliative, and sometimes rapidly curative, remedies. Fifteen grs. of the strontium salt may be given three or four times a day, half an hour before eating, or tablets of the arseniate of soda ($\frac{1}{50}$ of a gr.) should be given during the period of gastric activity. We have obtained the

best results from a combination of codein, extract of coca, and extract of hyoscyamus.

The intragastric douche may be of some service, and several glasses of hot water, given in the same manner as in the treatment of chronic gastritis, are sometimes beneficial.

The diet is determined in part by the associated disease, and should be sufficient to supply the needs of nutrition. Milk, finely-divided cereals, lean fish, the soft part of small, fresh oysters, the least excitant meats, dry toast, indifferently acting vegetables, etc., may be combined, and ordered in small and frequent meals. Sweets should be excluded, but enough fresh butter (unsalted) should be ordered to furnish the needed quantity of fat. The patient should live and exercise in the open air, and whenever subnutrition exists a very nourishing diet is absolutely essential.

II. ACORIA.

Acoria (*a*, privative, and *κορέννυμι*, I have enough; or *κόρος*, satiety) is a rare nervous affection of the stomach, characterized by loss of the special sensation of satiety. The patient never feels that he has eaten enough, whether the meal be small or very large. The appetite is no sharper than in health, indeed, it is often diminished, and the proper quantity of food to be eaten in order to avoid overloading the stomach must be estimated by reasoning.

Nature and Causation.—Some authors claim that the affection is a peripheral anesthesia, while others, whose views are upheld by autopsies, have found the trouble to be due to compression of the pneumogastrics and to softening of the nuclei of origin of their posterior roots. Acoria may be encountered in diabetes, but the large majority of cases occur in the neuropath, particularly after shock and depressing emotions (neurasthenia and hysteria). It may be produced by the crushing influence of a great sorrow. Acoria is an asthenic affection, and it is never associated with bulimia.

The sensation of satiety is not a mere negative sensation, as some contend, signifying the entrance of the hunger-center into a state of repose Acoria may coexist with anorexia. Neither is acoria a mere continuance of the hunger-center in a state of excitation, for the desire for food may, in this affection, disappear during the course of a meal in spite of the fact that the patient does not feel that he has had enough. Some of the patients with acoria have no sensation of fullness and

weight from overloading the stomach with food or from strongly inflating it with air, but others complain of pain as well as fullness and heaviness in the abdomen. In one of our cases we were able to locate these latter sensations in the colon. It seems probable that the sensation of satiety is identical with a particular state of the gastric "muscular sense," and may be intensified by certain associated sensations, as weight, fullness, discomfort, and cessation of the appetite. Acoria, in keeping with this view, is a special gastric anesthesia, which may be central or peripheral in origin.

Diagnosis.—The diagnosis consists in the detection of the symptom, and, when possible, also of its cause, the etiology serving as a guiding thread in the search. The only manifestation of acoria is the loss of the sensation of satiety, and the patient is exposed to myasthenia and gastritis from overeating.

Treatment.—The treatment of acoria consists chiefly in moral management, hygiene, and excitant hydrotherapy. The intragastric cold douche may be tried, and the hot or cold needle-bath should be used to tone the nervous system. Galvanization of the vagosympathetic, the cathode over the stomach, and the anode moved over the cervical centers, in the manner described in the chapter on electric treatment, should be tried. Less active are intragastric and epigastric electrization. Strychnin is valuable, and it should be given in increasing doses to the full physiological effect, and then continued for some time in ordinary doses. Frequent and small meals should be given, for the anesthesia is not likely to be relieved by overdistending the stomach. The treatment must also be causative, and a systematic cure may be required by the neurasthenia or by the hysteria which so frequently accompanies acoria.

III. PAROREXIA.

Parorexia (*παρά*, aside, and *ὄρεξις*, appetite) is a nervous perversion of the appetite. Bulimia, anorexia, and acoria are quantitative variations of the special sensations of the stomach; but parorexia is qualitative.

The appetite may be selective, and there may be a craving for special articles of the ordinary diet—pickles, sweets, ices, spices, condiments (malacia). This common perversion hardly deserves consideration in itself, unless accompanied by exclusion of the more nutritive foods; but it may play an im-

portant part in the etiology of the diseases of the stomach. The appetite may be more seriously perverted, and non-alimentary, injurious, even disgusting articles may be eaten (pica). This form is common among the little plantation negroes of the South,—the so-called "dirt-eaters,"—and seems to be more prevalent in the spring and in malarial regions. It may be associated with bulimia, and the immediate prevention of the pernicious habit may cause great suffering, which is relieved by the administration of food. It is a popular belief that "dirt-eating" is a sign of intestinal worms, and the association often exists. The habit may seriously affect digestion and nutrition, and is a cause of gastro-enteritis.

Parorexia is most common in the chlorotic or neurotic girl, particularly during the menstrual period. It frequently accompanies pregnancy, and may be caused by sexual excesses.

The insane and the hysteric may swallow all sorts of things—pins, needles, urine, feces; but this is a disease of the mind, and not a perversion of the appetite.

Treatment.—It is difficult to formulate a general treatment of parorexia. It is an irritative affection, and sedation is the controlling indication, which should be combined with good digestive hygiene and with the management of the associated condition. The stomach of the "dirt-eater" should be thoroughly washed out, and the complicating excessive secretion, bulimia, hypersthenic gastritis, malaria, intestinal worms, enteritis, anemia, or inanition should be given proper attention.

IV. ANOREXIA NERVOSA.

Complete loss of appetite may be a symptom of a large number of diseases, and does not constitute anorexia nervosa, which is a very serious nervous affection of the stomach. The appetite in this disease may be completely lost, but the loss is associated with a systematic refusal to take food for a particular reason or motive. The mind is up in arms, and with rare composure excludes all but a little food, and seems satisfied with the result. The loss of appetite is the only gastric symptom. The other symptoms are mental and those that result from the voluntary starvation.

Etiology.—Anorexia nervosa is a disease of the adult neuropath, but it may also develop in persons who are neither neurotic nor nervous. It is most frequent between fifteen

and twenty, and cases are very rare before this age and after thirty. Most commonly met with in young girls; it may also occur in young men. It is almost unknown in the active, outdoor-living country girl, and develops more frequently in the nervous, delicately constituted girl who is brought under the withering influences of enforced culture and imprisoned inactivity. It is sometimes, but rarely, engrafted on a painful affection of the stomach, and is frequently associated with displacements of the abdominal viscera; but no causal relation of this kind can be established, and the association is accidental, the deforming effects of the corset being very common.

The insane sometimes persistently refuse to take food; and hysteria seems able to mimic everything, and includes anorexia. This is the symptomatic form.

The primary form has been attributed to anesthesia, or to perversion of the sensibility of the gastric nerves (Fenwick), which prevents the starving tissues from giving expression to their need of nutriment. This theory is plausible, and the condition seems almost essential to the successful and complacent carrying out in practice of the steadfast and wilful refusal to take sufficient nourishment.

The adoption of a very insufficient diet may be self-imposed for a variety of reasons. The developing distaste for food is encouraged, and one article after another is excluded "because I do not like it," or "because this food increases my discomfort, and it is useless and harmful to eat what you can not digest." In some cases the conduct resembles closely hysterical posing, and is a plea for sympathy and for attention. The solicitude of family and friends only encourages the patient to persist in the voluntary starvation. Shock, grief, great sorrow, disappointment in love, and all causes of moral depression may mark the beginning of the disease. In many cases there is no explanation of the affection to be found in the habits, the circumstances, or the surroundings.

Clinical Description.—The clinical history of anorexia nervosa is that of slow, progressive inanition occurring under peculiar circumstances. In the beginning, digestive power is normal, and the insufficient diet is not enforced by a digestive disease but is adopted and maintained for certain motives or reasons known to the patient.

In the early stage, for a short while, the weight and the strength may be preserved. Great activity is sometimes shown by patients in order to prove the correctness of their conduct, and they often show a peculiar nervous restlessness.

The loss of color and the appearance of ill health may be hardly noticeable. This stage lasts longer in hysteria, of which the anorexia nervosa may be monosymptomatic, and seems to be made possible by the diminished nutritive activity, which is common enough in hysteria.

The inanition progresses and becomes more evident—in diminution of strength and weight, in the slow pulse, the cold extremities, constipation, scant and cloudy urine. The sleep is restless, disturbed by dreams, and there is commonly insomnia. The ankles may swell, the patient grows weaker, and excessive activity produces exhaustion. The family and friends become alarmed; but the patient, irritable and moody, is satisfied, and makes no effort to take more food, which, if forcibly or authoritatively given, may be rejected by vomiting.

If the slow inanition is not arrested, the condition becomes more and more serious. Pale, emaciated, weak, haggard, the patient stands on the verge of exhaustion, which is the second stage. The temperature is usually subnormal, but may be normal, or there may be at times slight fever. Inanition-delirium, with hallucinations, develops. All food may be refused. The digestive power is correspondingly depressed and the stomach may be intolerant. Constipation is obstinate, and the hard, lumpy stool consists of the secretions of the digestive tube. There may be periodical diarrhea, accompanied by the discharge of false membrane. The abdomen is sunken and the anterior abdominal wall is nearly in contact with the vertebral column when the patient lies on the back. The skin is rough, dry, and hangs like a bag around the flabby muscles and the prominent bones. The features are wasted, and the sunken eyes, with dilated pupils, are surrounded with dark rings. Bedsores develop, and the exhaustion may end in collapse, rapid fall of temperature, and death; or a fatal termination may occur earlier through pneumonia or some other complicating affection, the half-starved body falling an easy prey to bacterial invasion.

The urine diminishes in quantity, and in the stage of exhaustion is very scant, and only four to six gm. of urea are passed in the twenty-four hours. Albuminuria is exceedingly rare.

The blood is that characteristic of simple inanition. The percentage of red cells divided by the percentage of hemoglobin is equal to one The fresh specimen of the blood examined with the microscope may be much better than the appearance of the patient would lead one to expect. The number of red corpuscles in the cubic millimeter may be nearly normal, the volume of the blood being probably

diminished, and this vital fluid suffers only after most of the stored fat and muscles have been eaten away. The number of white corpuscles in the cubic millimeter of blood is very small, falling as low as 2000.

Diagnosis.—A complete loss of appetite, the absence of any serious organic disease, the mental condition, the characteristic genesis and evolution, the slow progressive inanition due to an insufficient diet,—leave those who have seen the disease in little doubt as to its nature.

Differential Diagnosis.—A large number of diseases are accompanied by loss of appetite and by emaciation. The appetite is diminished or lost in most of the febrile diseases. Diseases of the blood, cachectic conditions, severe intestinal diseases, the painful diseases of the abdomen, chronic malaria, and some cases of tuberculosis, are accompanied by loss of appetite. Opium, alcohol, tobacco, digitalis, and many other drugs, when used for a long time, destroy the appetite. Chronic asthenic gastritis, gastric retention and carcinoma are usually accompanied by loss of appetite; but in all these diseases the patients are alarmed at their condition, and are willing to eat, and do all in their power to get well. The causative disease will be discovered on examination. When the loss of appetite is symptomatic, symptoms are present which do not belong to the symptom-group of anorexia nervosa, the blood and urine changes of which may also be valuable differential signs. In the early stage, the gastric functions will be found normal, and even in the stage of exhaustion the digestive power may be good. There are no functional signs of an organic or functional disease of the stomach.

Tubercular meningitis and tubercular peritonitis may be difficult to exclude. The somnolence, and the delirium in the advanced stage, if the patient be then seen for the first time, and the progressive character of the trouble, may well excite suspicion. But other signs of these diseases may be present, and the wilful and dogged refusal of food, in spite of the integrity of gastric digestion, is characteristic of anorexia nervosa.

Treatment.—The indications in the management of a case of anorexia nervosa are: (1) To restore the balance of nutrition; (2) to treat the gross symptoms; (3) to improve the mental and nervous condition.

In the early stage it is difficult to secure the co-operation of the family and friends, who often fail to see the necessity of firm and absolute control of the patient. The digestive

power is still good, and in order to meet the first indication it is only necessary that a sufficient quantity of food be given. To do this, while surrounded by the family and friends, may tax all the resources of the physician.

It is unnecessary to prescribe a restrictive or exclusive diet. The articles may be selected from the diet of health, and the taste of the patient should be taken into consideration. But the physician who accepts suggestions runs the risk of losing his authority, and without moral control his efforts will prove useless. A bitter tonic may be given,—strychnin, quinin, columbo, etc.,—but this alone will do little good. Fowler's solution combined with an aromatic bitter may be tried; or the interior of the stomach may be douched with a ½ per cent. salt solution or with a bitter infusion (Kussmaul, Fleiner). Penzoldt recommends orexinum basicum (four grs. in a capsule or wafer at 11 A. M., with a cup of bouillon). The constipation will disappear with the increased amount of food; and warm clothing, moderate exercise, hydrotherapy, and other measures to tone the nervous system should not be neglected. The physician must assume complete control, and regulate minutely the mode of life, and strictly enforce a sufficient diet and proper hygiene.

In the advanced stage the patient should be isolated, and given a gentle but firm nurse. If the stomach is intolerant, rectal nourishment should be used; and milk, eggs, and meat juices, etc., should be given by the mouth as soon as the vomiting, which is a rare complication, is under control. Absolute rest in a warm bed, systematic feeding, using authority rather than the stomach-tube, massage, electricity, hydrotherapy, are the elements of the combined rest-cure. Few symptoms require drugs for their control, and it is a bad plan to get these patients accustomed to their employment and dependent upon them. Supplementary chemical treatment with pepsin and with hydrochloric acid may be of some aid in securing the digestion of a sufficient quantity of food in the beginning of the treatment.

V. GASTRALGIA NERVOSA.

Gastralgia is an intermittent, painful affection of the stomach, occurring in paroxysms independent of the period of functional activity or of the existence of another disease of the stomach. It is in its nature the gastric cry of an irritated nerve-ending or nerve-center—a pneumogastric neuralgia.

Etiology.—Paroxysmal gastric pain may be symptomatic, reflex, central, or neuralgic.

Symptomatic paroxysms of gastric pain occur in ulcer of the stomach, in cancer, in obstructive and myasthenic retention, in acute (toxic or mycotic) and chronic (hypersthenic and atrophic) gastritis, in adenohypersthenia gastrica, in gastric spasm and spasm of the orifices, in perigastritis and perigastric adhesions, and in gastroptosis. The paroxysms of pain associated with these diseases should not be confounded with gastralgia nervosa, nor should painful gastric cramps be described, as is done by some authors, under the heading "gastralgia," which is a neuralgia of the stomach.

Reflex gastric pain is usually spasmodic and not neuralgic. Gastralgia, however, is very seldom caused reflexly by diseases of the generative organs, of the liver, of the spleen, of the pancreas, and of the bladder.

Painful gastric crisis may be produced by diseases involving the nuclei of origin of the pneumogastrics (tabes, multiple sclerosis, myelitis, and tumors), by compression of the trunks of the pneumogastric nerves, and by irritation of the peripheral endings of the pneumogastrics or of the sympathetic ganglia.

Idiopathic gastralgia nervosa is not a frequent disease, and is due to the same causes as other neuralgias. Neurasthenia, malaria, syphilis, chlorosis, oligocythemia, uricemia, and autointoxication are the most common causes, and masturbation and sexual excesses are often associated, seemingly in a causal relation, with the trouble. Chlorosis and oligocythemia frequently cause adenohypersthenia gastrica, sometimes hyperesthesia, and, rarely, gastralgia. The abuse of tobacco is responsible for some of the cases in men, but gastralgia nervosa is most common in women between the fifteenth and thirtieth years who belong to neuropathic families. It sometimes follows typhoid fever and influenza. Malarial gastralgia may be periodical, accompanying the chill or taking its place.

Clinical Description.—At the bedside, gastralgia is easily separable into two forms—the mild and the severe. In the mild form the pain is bearable, closely limited to the region of the stomach, and of short duration. There is little or no prodromal nausea or salivation, but only a slight feeling of distention, to usher in the attack. There is no fermentation, and the variations of secretion do not get beyond the normal limits. There may be a small quantity of gastric juice containing a trace of free hydrochloric acid in the stomach, if the attack should occur during the period of gastric repose;

and its presence may be best explained as the result of the vasomotor dilatation that is frequent in parts affected by neuralgia. During the attack the stomach is but little distended or contracted, and the organ, apart from a slight sensitiveness on pressure, yields no abnormal physical signs. The attack is usually unassociated with heartburn, or pyrosis, and ends with a moderate sensation of hunger, slight drowsiness, and a free discharge of clear urine of low specific gravity, of slight acidity, and which precipitates the earthy phosphates on heating. This is the mild form.

The duration of the mild attacks is short—seldom longer than a few hours. The pain is often moderated by strong pressure over a large area of the epigastrium, and it may be rapidly and completely relieved by the pure sedative influence of anodal galvanization.

The paroxysms of the severe form usually begin with a well-marked sensation of distention of the stomach and with loss of appetite. The beginning is violent, sudden, without adequate local cause, and independent of the functional activity of the stomach. Then the severe, tearing, cutting, throbbing, aching, or burning pain appears in the epigastrium and radiates through the abdomen, behind into the back, along the intercostal nerves, and under the sternum into the lower part of the esophagus. The irradiated pains may be very severe and accompanied by a pinched, anxious, perspiring face, cold extremities, retracted abdomen, and a small, jerky pulse, usually frequent, sometimes slow, with pulsation of the abdominal aorta. Or the patient may be doubled together with contracted abdominal muscles, the breathing being thoracic and shallow. After a variable duration the pain subsides, and the exhausted patient may fall asleep. During the attack the urine is of high specific gravity and but little is secreted. The paroxysm may last several hours and may end in vomiting; or the pain may gradually cease with the discharge of a large quantity of clear, or sometimes slightly clouded, urine of the same character as in the mild attack.

The violent attacks may last one or two days, or only a few hours, and may recur after long, short, or irregular intervals. The pain is calmed by neither alkalies nor albuminous food, and the paroxysms occur regardless of the functional activity or repose of the stomach. If they occur during the period of digestion, the attack may end with critical vomiting; but the attacks recur in relation with the exacerbations of the causative disease and are most frequent after cerebral or physical fatigue.

Diagnosis.—Intermittent paroxysmal attacks of gastric pain, beginning suddenly, becoming rapidly intense, in no constant relation with the taking of food or with the evolution of gastric digestion, unassociated with abnormal functional and bacteriological signs, occurring in the absence of any other disease of the stomach, separated by intervals of normal painless digestion, are so characteristic that the diagnosis may be easily made. But the etiological diagnosis may present great difficulties, and a careful search should be made, and the diseases mentioned under etiology should be excluded, before the gastralgia is pronounced to be primary and idiopathic—a form which close study will prove to be rare.

Differential Diagnosis.—The differentiation of gastralgia from other painful paroxysmal diseases must often be made by exclusion. It may be confounded with some of the painful gastric diseases or with painful diseases of other abdominal organs. It may also be confounded with intercostal neuralgia and rheumatism of the abdominal muscles.

Intercostal neuralgia may be due to the same causes as gastralgia, and is frequent in anemic girls, and is most common on the left side. The intercostal neuralgic pain may be severe and may resemble in location and intensity that of gastralgia. The neuralgia of the lower intercostal nerves may be concentrated in the epigastrium. The area of the radiations is different; the patient locates the intercostal pain in the thoracic and the abdominal walls, and the whole course, or special points of the intercostal nerve, or nerves, are painful on pressure. The intercostal neuralgia, being superficial, is more readily relieved by sedative galvanization. The intercostal pains of spinal disease are bilateral and constricting, and are associated with the special signs of the spinal trouble. But it should not be forgotten that the pain of gastralgia may radiate along the intercostal nerves.

Myalgia may be localized in the epigastrium and the left hypochondrium. In myalgia and gastralgia the abdominal muscles are contracted, and the pain may be paroxysmal and similar in location. The myalgia is relieved or increased, respectively, by relaxation or contraction of the affected muscles. The painful area corresponds with the location of the muscles, and the extension of the trouble to the lumbar or other muscles outside of the gastric area would be a distinctive characteristic. Muscular rheumatism, as Leube has shown, may be accompanied by fever, and there is no increase of temperature in gastralgia.

Gall-stone colic, which may be itself the cause of gastralgic

crises, probably through irritation of the solar plexus, may be mistaken for gastralgia. In cholelithiasis the pain is to the right of the median line; radiates sometimes also to the right and to the region of the right scapula; a painful pressure-point to the right of the twelfth dorsal vertebra is common; the liver may be enlarged and tender; the epigastric tenderness corresponds to the area and the form of the left lobe; the gall-bladder may be distended, and may present a visible and palpable tumor, and give rise to friction-sounds during the movements of the diaphragm; there may be jaundice and fever, and gall-stones may be found in the feces. The attacks of hepatic colic are often closely related to dietetic excesses. In atypical cases all these differential characters may be absent, and a seemingly typical gastralgia may later prove to be a no less typical gall-stone colic.

Intestinal colic is not likely to be be mistaken for gastralgia, except when located in the transverse colon. There may be signs of chronic lead-poisoning or a history of constipation. The abdomen is fuller than in gastralgia; the intestine can be felt distended with gas, or it rolls like a hard cord under the finger. The radiation is into the lumbar region or over the sacrum, and the paroxysm ends with the evacuation of the bowel, which often contains mucous shreds or hydrosulphuric acid gas. The intestinal pain is also distinctly peristaltic, or spasmodic, and the point of the greatest intensity follows the course of the bowel.

Hyperesthesia gastrica is easily differentiated from gastralgia. The pain of hyperesthesia is not spontaneous, is excited immediately by the contact of the ingested food, and it subsides with the evacuation of the stomach.

Adenohypersthenia gastrica and chronic hypersthenic gastritis are frequently mistaken for gastralgia. The hyperchlorhydria, or hyperchylia, at once excludes gastralgia. The paroxysms begin and develop more slowly, after the ingestion of food, and in close relation with the evolution of secretion. The pain is moderated by alkalies, by water, and by albuminous food, and it is digestive. The functional signs, however, in all doubtful cases, are absolutely conclusive, for hyperchylia and hyperchlorhydria exclude gastralgia. Paroxysmal pain characterizes a clinical form of chronic atrophic gastritis; but the achylia, which always accompanies this disease, is never found in gastralgia.

The differentiation of atypical ulcer and gastralgia may be difficult or impossible. The typical, painful paroxysms of ulcer, excited by food and digestion, and relieved by the evacuation of the stomach, present no difficulty. Neither can

there be any doubt when there are strictly circumscribed and painful epigastric and dorsal pressure-points, and when the pain is increased by the movements of the body and is relieved by rest in a particular position. But the ulcer may be atypical in its expression, and may leave the physician in doubt after all the etiological, subjective, and objective signs have been weighed. In the absence of the possible conclusive functional signs, the case should be treated as ulcer until more light can be obtained. Such a course protects the physician and gives the patient the benefit of all doubt.

Epigastric pain may be caused by small herniæ in this region, by cysts or carcinoma of the pancreas, by pancreatitis, by pancreatic calculi, by duodenal ulcer, and by perigastric adhesions. When the pain is due to adhesions of the stomach, it recurs with greater regularity than in gastralgia, and it may be produced by inflating the stomach or by overloading it with food.

Treatment.—The treatment of gastralgia is etiological and symptomatic. To relieve the pain, anodyne and sedative treatment is indicated. In the mild form, ten grains of antipyrin often promptly relieve the pain. If the pain be very severe, it is best to give at once the sovereign remedy, which is a preparation of opium,—morphin or codein,—administered hypodermically or by the mouth. Hot poultices (or a hot-water coil over wet flannel) should be placed over the stomach, and hot drinks should be given by mouth. The deodorized tincture of opium is also a good preparation. Belladonna, Hoffman's anodyne, and chloroform water have also been recommended, but are not trustworthy. In the recurring mild attacks, aconite and gelsemium may prove serviceable, and act best when given simultaneously in small, repeated doses. We usually order a combination of codein ($\frac{1}{4}$ of a gr.), ext. cannabis indica ($\frac{1}{10}$ of a gr.), atropin ($\frac{1}{200}$ of a gr.), and aconitia ($\frac{1}{400}$ of a gr.). If malaria be the cause, the attacks of gastralgia are periodical. Quinin cures these cases promptly. We prefer the muriate when the drug is given by mouth, and it should be administered in a single dose (10 to 15 grs.), five hours before the time for the recurrence of the attack. Or the quinin may be given by rectum, the following formula being a good one:

℞. Quininæ muriat., ... grs. x–xx
Morphinæ muriat., ... gr. ⅛–¼
Sodii chloridi, ... grs. v
Aq. amyli, ... ℥ij.
M. et ft. sol.
Sig.—Inject slowly into the rectum three hours before the attack.

Or the bimuriate of quinin (grs. xxx to aq. dist. ℥j) may be given hypodermically, the muriate of morphia being combined with the quinin if it be desirable. Administer 15 to 20 minims two hours before the time for the attack. It is our custom to combine with the quinin, during the time of the administration of full doses (three to five days), either morphin or codein, which act as synergists.

Galvanization often gives prompt relief, and is indicated where it is advisable to avoid the use of morphin. The anode (100 sq. cm.) is placed over the stomach, and the cathode, of the same size, is held to the left of the dorsal spine. A current of three to five milliamperes is slowly and gradually turned on, is allowed to flow from five to ten minutes (until the pain ceases), and is slowly reduced to zero before removing the electrodes, in order to obtain the pure sedative polar influence of the anode. Intragastric galvanism may be tried by those who prefer this method of using it. It matters little whether intragastric or epigastric galvanism is employed, provided it be administered so as to get a sedative influence. During the intervals, sedative galvanism and aconitia give excellent results, and the special indications furnished by the etiology of the neuralgia should be met—forbidding the use of tobacco and sexual excesses, building up the blood and nutrition and the nervous system, curing the malaria, and preventing auto-intoxication.

VI. HYPERESTHESIA GASTRICA.

Like gastralgia, hyperesthesia may be only a symptom or it may be a primary affection of the stomach. In health a person is unconscious of the contact of the normal contents of the stomach with the gastric mucous membrane. In simple hyperesthesia gastrica the mucous membrane is excessively sensitive without the presence of either an anatomical lesion or of a disorder of secretion. The patient becomes conscious, often painfully conscious, of the contact impressions, which in health are unperceived. The disease is analogous to hyperesthesia of the skin, and differs essentially from gastralgia, which is a pneumogastric neuralgia.

Etiology.—Hyperesthesia gastrica may be a symptom of ulcer and of both adenohypersthenia gastrica and chronic hypersthenic gastritis. As a distinct, unassociated affection of the stomach it occurs in diseases of the central nervous

system, in chlorosis, in anemia, in the arthritic, and in the neuropath. Gastric hyperesthesia may be the first and only symptom of uremia. It may be a monosymptom of hysteria or may coexist with other manifestations of this psychosis. It may be excited by irritant foods and drinks, by tea, coffee, tobacco, excesses in venery, onanism, and masturbation. It is a common sign of narcotic drug habits, and may be produced by chloroform narcosis. It is a symptom of absinthe alcoholism, is not rare during convalescence from exhausting diseases, sometimes precedes and sometimes follows gastralgia nervosa, and may be induced by prolonged fasting and by the protracted use of an exclusive and insufficient diet. Hyperesthesia gastrica is a common result of an insufficient or exclusive diet, but the basis of the trouble is most frequently a neuropathic or an arthritic soil.

Clinical Description.—In the mild form, immediately after the introduction of food there is a peculiar uneasiness and discomfort, a feeling of local irritation, and tingling, shooting pains. These symptoms continue throughout the period of digestion, and disappear with the evacuation of the stomach.

In the well-developed form the contact of food produces immediate pain, which, when severe, may excite nausea and vomiting. The symptoms are excited alike by fluids and by solids; indeed, fluids containing an excitant seem to produce a more diffused pain. The pain, often preceded and accompanied by a sensation of weight and fullness, continues throughout the period of gastric activity; and nausea, a sensation of cold and heat, conscious gastric arterial pulsation, and vomiting convince the patient of the existence of a very serious organic disease. When water is introduced through the tube into the stomach, retching and vomiting are immediately and almost invariably excited. Through fear of pain, one article of food after another is refused; through vomiting the supply of nutriment may be reduced below the needs of nutrition, and emaciation may become progressive. There may be no emaciation, or there may be emaciation proportionate to the quantity of food lost by vomiting and to the insufficiency of the diet. Gastric hyperesthesia is the forerunner of anorexia nervosa and of habitual vomiting.

During the period of gastric repose there is often a sensation of emptiness, often decidedly unnerving and unbearable, and associated with slight vertigo and faintness. A peculiar form of hyperesthesia gastrica is manifested by short, painful paroxysms (fifteen to thirty minutes), which occur as soon as the stomach becomes empty. The pain may be relieved by

food, but it is not relieved by soda, the sodium chlorid formed increasing the pain and often exciting nausea. It is probable that the pain is produced by the action of free HCl on the oversensitive gastric mucosa, secretion continuing after the evacuation of the contents of the stomach into the duodenum. In some cases sensation is dissociated, either in the periphery or in consciousness. There may be a morbid sensibility to slight changes in the temperature of the food, the painful impression of contact being momentarily less pronounced; or the gastric contents may produce now a sensation of cold, now burning, now pain, and these sensations are sometimes spontaneous and perceived when the stomach is empty.

In hyperesthesia gastrica the skin over the epigastrium is often very sensitive. Deep pressure reveals the morbid sensibility of the whole stomach, there being no circumscribed painful points. The area of diffused tenderness is sharply limited and corresponds closely with the size and form of the stomach. By moderate vertical pressure with the fingers the size and position of the stomach may frequently be determined with exactness.

The secretory function of the stomach is normal. The contents, after the test-breakfast, reveal normal chemical, microscopical, and bacteriological signs. There may be inconstantly a small excess or a slight diminution of free HCl, which may be plausibly explained by the accompanying vasodilatation.

Diagnosis.—The pain due to contact, and occurring immediately and invariably after the ingestion of food, or as soon as the stomach is empty, and with the associations detailed in the clinical history, and with the normal functional signs, leave little room for doubt as to the nature of the trouble. The etiology may also aid in the diagnosis. The pain is excited by the contents of the stomach—the food, digestive products, and the free hydrochloric acid. Consequently the symptoms are digestive, occur regularly, and for a number of days or weeks after meals, or at the moment when the stomach first becomes empty. Hyperesthesia gastrica, consequently, differs widely from the paroxysms of gastralgia nervosa.

Differential Diagnosis.—The only diseases likely to be confounded with hyperesthesia gastrica are atypical ulcer, adenohypersthenia gastrica, and hypersthenic gastritis. In adenohypersthenia and in the hypersthenic form or stage of gastritis, the pain does not occur so soon after the ingestion of food, and the stomach only becomes intolerant toward the height of secretion. The pain is also in strict relation

with the quality of the food, and is relieved by albuminous foods and by alkalies. In hyperesthesia the pain is immediate, and is due to contact and not to functional excitation, and is produced by all sorts of food. The abnormal chemical signs characteristic of functional and organic adenohypersthenia are absent.

Ulcer may be manifested only by pain, but the ulcer pain has distinctive features. Cases where the pain does not occur immediately after taking food present no difficulty. But hyperesthesia may be a complication of ulcer. Even in the absence of the characteristic relations of the pain of ulcer to the quality of the food, to the evolution of secretion, to the movements and repose of the body, and even in the absence of the characteristic circumscribed epigastric and dorsal pressure-points the abnormal functional signs of ulcer would still be conclusive. Both diseases are frequent in the same class of patients, and if there is doubt after weighing all the signs, the case should be treated as ulcer.

Treatment.—It is best to put these patients at once to bed. The more absolute the repose of body and mind, the more rapid will be the cure. Sedative galvanization, applied as in the treatment of gastralgia, but with the current of very low density—as two milliamperes with an electrode 100 sq. cm., daily sittings. A cold compress (Winternitz's compress is the best) should be kept constantly over the abdomen. Nitrate of silver is the best medicine, and often acts with remarkable efficiency. One grain shouldbe dissolved in two ounces of distilled water, and one or two teaspoonsful should be given three times a day on an empty stomach. It is often unnecessary to continue the drug longer than two or three days. A more efficient method is to spray or douche the stomach with a solution of nitrate of silver. In our experience, the bromids do no good.

The diet should be non-exciting and indifferent in its physiological action on the stomach. For a short period a pure milk diet or milk and lime-water should be employed; as soon as the pain is controlled, cereal gruels, soft-boiled or poached eggs, and meats, vegetables, and other foods should be added, in the order given in the chapter on Diet (Section III, chap. II). Or it may be best to employ exclusive rectal feeding for a few days.

The etiological treatment will demand attention, and consists in medication directed against the hysteria, anemia, chlorosis, neuropathic or arthritic constitution, and the correction of excesses and of bad habits.

CHAPTER II.

THE DYNAMIC AFFECTIONS OF SECRETION.

Some authors deny the existence of persistent disorders of secretion which are not produced by an anatomical lesion of the gastric mucosa, and the majority of writers consider all the dynamic affections of secretion to be neuroses. In our opinion both these contentions are erroneous. There is no doubt that persistent abnormalities of secretion are frequently functional signs of the anatomical diseases of the stomach. No one will deny that secretion may be disturbed through the nerves which control it. Prolonged study and careful investigation have led us irresistibly to the conclusion that the disorders of secretion are not always due to an alteration of the mucosa or to a neurosis of the vagosympathetic system.

It is, furthermore, the custom to describe a very special condition of the stomach as a distinct disorder of secretion, or neurosis of the stomach. This condition has been named "continuous secretion," or gastrosuccorrhea, or Reichmann's disease, and it may be either periodical (gastrosuccorrhea periodica) or continuous (gastrosuccorrhea continua chronica). We are unable to convince ourselves that continuous gastrosuccorrhea exists as a dynamic affection. This condition of secretion may be met with as an episode in obstruction of the pylorus, in myasthenia with secretory irritation due to the retention of the contents of the stomach, or in the hypersthenic form of chronic gastritis. It is not a distinct morbid entity, but is a special symptom—a mere complicating condition. Continuous secretion, in our opinion, is not a severe form of supersecretion without an anatomical lesion of the mucous membrane, nor is it a delayed reaction of the secretory nerves to the excitation produced by the meals. The normal stomach does not secrete when it is empty, and, at most, only 10 to 20 c.c. of gastric contents can be removed when the tube is introduced into the normal stomach in the early morning before breakfast; but sometimes when the tube is introduced at this time (even when the stomach has been thoroughly washed out the evening before and left empty) it will be found that the stomach contains much more than 20 c.c. of a liquid rich in hydrochloric acid

and the digestive ferments. This is considered the characteristic functional sign of continuous secretion. The sign exists, but we maintain that its interpretation is wrong. The secretion has merely accumulated in the stomach (myasthenia, pyloric obstruction), or it has been formed as a result of hypersthenic gastritis, or, in case the stomach has not been washed out, it may have been excited by retained food and digestive products. The evening lavage, or lavage followed by a few days of rectal feeding, or the water test, considered in combination with the clinical history and with the other objective signs, will reveal the cause of the gastrosuccorrhea and demonstrate its non-existence as a dynamic affection of the stomach. It is always due to retention, or to hypersthenic gastritis, or to both.

Unfortunately, the words in common usage are unsuitable for designating the dynamic affections of secretion. Superacidity (or the mongrel word, hyperacidity) denotes abnormal increase of the acidity of the contents, whether it be due to hydrochloric or to organic acids. Hydrochloric superacidity is more exact, but the term is also used to denote the functional sign of certain anatomical diseases of the stomach. The same objections apply to subacidity. Supersecretion denotes an increase of the quantity of secretion, whether it be a particular morbid process or a functional sign. Hyperchlorhydria and hypochlorhydria denote a pathological increase or decrease of hydrochloric acid in the filtrate of the gastric contents at the acme of digestion, and do not embrace all the secretory disturbances which are found in the two classes of the dynamic affections. In order to avoid confusion and to embody an exact conception of their nature, we shall divide the dynamic affections of the stomach into two large classes, and describe them, in keeping with references in other parts of this book, as adenohypersthenia gastrica (*ἀδήν*, gland, *ὑπέρ*, excessive, and *σθένος*, strength; excessive glandular activity) and adenasthenia (*ἀδήν*, *α*, and *σθένος*) gastrica. These terms denote the morbid processes which are manifested by the disturbances of secretion, and stand in clear relief with the similarly formed terms, myasthenia gastrica and neurasthenia gastrica.

1. ADENOHYPERSTHENIA GASTRICA.

Adenohypersthenia gastrica is a dynamic affection characterized by the secretion of a gastric juice which is abnormally

rich in hydrochloric acid or which is excessive in quantity. The hyperchlorhydria (ὑπέρ, χλωρός, and ὕδωρ) and the hyperchylia (ὑπέρ, excess, and χυλός, juice) gastrica are due neither to an anatomical lesion of the mucosa nor to motor insufficiency. Achylia (a lack of juice) gastrica has recently been employed by Einhorn to denote a permanent absence of gastric secretion, and the similarly formed terms, hyperchylia gastrica and hypochylia gastrica, may be used to denote a pathological increase or decrease of gastric secretion. Hyperchylia gastrica is here employed to designate the dynamic affection of the stomach which is characterized by supersecretion.

Hyperchlorhydria and hyperchylia gastrica are closely related in their etiology, and hyperchlorhydria may be the forerunner of hyperchylia; but this does not establish the identity of the two affections. The one may be a sequel of the other, but hyperchylia gastrica may be milder than the severe cases of hyperchlorhydria, and may not be accompanied at the acme of the digestion of a test-meal by hydrochloric superacidity. The one is only a qualitative modification of secretion, and the other is essentially a quantitative disturbance. The two affections differ in their subjective manifestations, in their physical, functional, and bacteriological signs, and in their treatment. Hyperchlorhydria is always digestive; hyperchylia gastrica may be digestive or paroxysmal.

(A) HYPERCHLORHYDRIA.

The causes of hyperchlorhydria are those common to a large number of other diseases of the stomach, and are as often found in the constitution and temperament as in the mode of life and the alimentation. The abuse of condiments, the eating of large quantities of red meats, and imperfect mastication are common causes. The disease is most frequent in youth and manhood, most of the cases occurring between the ages of fifteen and forty. Sex seems to be without influence. Like other dynamic affections, it is most frequent in the arthritic and the neuropath: in neurasthenia, in hysteria, and in melancholia. According to our observation, it is quite frequently associated with intestinal auto-intoxication. It is common in cholelithiasis, in renal lithiasis, in chlorosis, and in chronic tobacco-poisoning. It is frequent in chronic malaria, even before quinin has been taken.

Mental and moral causes play an important part. Cerebral fatigue may mark the beginning of the trouble, and illustrates

the close relations existing between the brain and the abdominal sympathetic, on which Leven has laid so much stress. It is very frequent among students. Prolonged excitement, worry, and excesses of all sorts are other causes. There is no distinct relation between the disorder of secretion and the nature of the cause.

Clinical Description.—Hyperchlorhydria may be latent, resembling in this respect many other diseases of the stomach, or the subjective manifestations may occur intermittently, in spite of the unbroken continuity of the digestive secretory irritation. The trouble may begin suddenly after a particular meal; or it may develop more gradually, a meal composed chiefly of starches, cereals, sweets, vegetables, fruits, and fat causing discomfort and pain at the height of gastric digestion.

The symptoms are digestive, and are in strict relation to the evolution of secretion. In a mild attack, which is the rule after a small meal, such as a breakfast composed of a cereal, eggs or meat, and *café au lait*, there is slight discomfort and uneasiness in the stomach, which usually begin with the appearance of free HCl in the contents; and, later, there may be acid eructations, heartburn, or even severe pain—all of which disappear with the evacuation of the stomach. The severe attacks, which occur chiefly after a meal the action of which is somewhat irritating and leaves a large quantity of hydrochloric acid free or which was eaten when tired, are accompanied by uneasiness, heartburn, eructations of a bitter, sour fluid, severe pain, and sometimes vomiting. The intensity of the symptoms keeps pace with the evolution of secretion and of digestion. The evacuation of the stomach marks the end of the attack. The pain is most intense during the course of digestion, when free HCl is greatest, and is temporarily relieved by nitrogenous food, and, more permanently, by a full dose of an alkali. The appetite is good and thirst is almost invariably intense. In some cases the local digestive symptoms are accompanied by the symptoms of general neurasthenia, which are most prominent during digestion. The disease is a predisposing cause of ulcer, and, unless arrested early, is likely to end in hypersthenic gastritis.

The subjective manifestations, which are exclusively digestive, may be continuous, remittent, or intermittent, with intervals during which digestion is painless. The digestive secretory irritation continues from day to day, with occasional exacerbations; but in the early period of the affection, under the influence of rest and a bland diet, secretion may intermittently become normal.

The functional signs are characteristic. The motor function is usually normal after a test-meal; after a large meal, accompanied by severe pain, pyloric spasm may delay evacuation. The motor function is excited by the excessively acid contents, and the stomach should become empty more rapidly than in health; but the hyperesthesia of the pylorus and of the duodenum may prevent the rapid evacuation by producing spasm of the pylorus. There is no morning splashing, and usually there is no splashing half an hour after drinking a glass of water on an empty stomach. If splashing can be elicited during the digestive period it is circumscribed, and never extends beyond the normal limits of the stomach. There is no motor insufficiency, except the occasional and slight stagnation which may result from pyloric spasm. After the water-test the stomach will be found empty within the normal period, and the stagnation, if it occurs, is, consequently, not due to myasthenia.

The resting stomach will be found empty. Albumins are digested with unusual rapidity. At the end of two hours the meat of the test-meal of Germain Sée is almost completely dissolved, and the few fibers found with the microscope are undergoing disintegration; propeptones are abundant, and the biuret and Almèn reactions for peptones are plainly positive. Starch is not so well digested as in health. The inhibition of salivary digestion occurs so much earlier than in health that Lugol's solution gives a blue or purplish-red coloration when added to the filtered contents. The contents obtained one hour after a test-breakfast are excessively acid (normal, 60), composed of both free (H) and (C) combined HCl. Free HCl appears much earlier (ten minutes) in the contents after the test-breakfast than in normal secretion (twenty to thirty minutes); and also about twice as early as in health after the Sée and after the Riegel test-dinners. Albumin digestion is very active and rapid, and combined HCl may be excessive, even though there should be no obstructive stagnation and consequent accumulation of digestive products. The hydrochloric acidity is excessive during the decline of digestion. Consequently secretion may be rapid, and very rich in both hydrochloric acid and digestive ferments. There are no organic acids, or only unimportant traces. The filtered contents are rich in ferments and possess very high digestive and milk-coagulating powers. The tube digestions are very rapid—the acidulated 50 per cent. dilution often digesting as rapidly as the filtrate of the normal contents. Labferment and labzymogen are both very active. The mucus, and the epithelium, and the germ

growth are not excessive nor abnormal. The tube fermentation tests are negative.

The epigastrium is painful on pressure during the digestive attacks, and more markedly so over the pylorus. The skin may be hyperesthetic. During the interval while the stomach is empty there is much less tenderness and hyperesthesia.

The urine formed during the period of gastric digestion is poor in chlorids, nearly neutral, and precipitates the earthy phosphates on heating, and sometimes on standing. The diminution of the acidity of the urine secreted during gastric digestion is a rough measure of the increase of the hydrochloric acidity of the gastric juice.

The bowels are constipated, but in spite of the excessive acidity of the chyme the starches and fats may not exist in abnormal quantity in the stools, and the balance of nutrition may be maintained. The weight, and the strength, and the color may be those of health, but moderate emaciation is frequent. The appetite is preserved, or it may be very sharp, and a meal taken later than usual will be preceded by hunger.

Diagnosis.—The diagnosis should present little difficulty if the examination has been complete. The following diagnostic signs should be clearly fixed in the mind:

The general health, the weight, and the strength are well preserved if the diet has not been restricted. The food being well digested, and not destroyed by fermentation, nor lost by vomiting or by diarrhea, there is no reason why the balance of nutrition should not be maintained. The good appetite will secure the ingestion of a sufficient quantity of food, unless the diet be reduced on account of fear of pain or in consequence of improper treatment.

The symptoms are all digestive and gastric. During the period of repose of the stomach there is no complaint. The symptoms do not begin immediately after the ingestion of food, but develop in relation with the evolution of secretion, and are most intense during the period of greatest free hydrochloric acidity. The severity of the attack is dependent upon the action of the food, upon the power which it has of combining the secreted HCl, upon the activity of hydrochloric secretion, and upon the irritability of the nervous system at the moment when the food is eaten. The pain is calmed by albuminous food and alkalies; it is uninfluenced or made worse by electricity, and disappears with the evacuation of the stomach. The symptoms may appear only after the chief meal. A glass of milk taken alone is usually rapidly and comfortably digested.

The objective signs are even more characteristic. The stomach is normal in position and in size, the motor function (unless there is spasm of the pylorus) and absorption are normal, and there are no bacteriological nor anatomical signs. The resting stomach is empty. During digestion an excessively acid gastric juice, rich in both ferments, is secreted.

Differential Diagnosis.—The differential diagnosis, in spite of the clear-cut features of the disease, may present some difficulties, and in some cases only a probable decision can be made.

Myasthenia with supersecretion presents a very similar group of symptoms, which are digestive, increase with the evolution of digestion, and may attain their climax with paroxysms of pain. The objective signs can alone differentiate the two diseases. These signs are those of myasthenia, which are never present in hyperchlorhydria. After the test-breakfast the quantity of contents is normal in hyperchlorhydria; the emulsion-meal of Mathieu shows that the contents are evacuated into the intestines with normal rapidity; after a Leube-Riegel or a Germain Sée meal the evacuation of the stomach is not delayed, and digestive products do not accumulate in the stomach. In myasthenia there is splashing when a glass of water has been given on an empty stomach, after the normal interval has elapsed; and the stomach during digestion is often flabby. Two glasses of water taken on an empty stomach are not evacuated within one and one-half hours, for myasthenia is a "dyspepsia of liquids." The motor insufficiency revealed by the water-test is the most characteristic sign, and the supersecretion increases or decreases with the increase or decrease of the myasthenia. The free HCl may appear early in myasthenia, but the acme of hydrochloric acidity is postponed, and the decline of secretion is delayed. If the myasthenia is associated with retention, the differentiation is easy. The digestive subjective symptoms, which in myasthenia with stagnation extend over a longer period than in hyperchlorhydria, become confused with the retention symptoms occurring during the period of normal gastric repose. The retention of food excludes hyperchlorhydria, in which affection there is never found in the stomach in the early morning before breakfast either retained food or accumulated digestive products, or an excessive quantity of gastric juice.

The displacements of the stomach may manifest the same subjective symptoms—uneasiness, acid eructations, heartburn, and gastric pain two to four hours after meals. The pain, however, is peristaltic, and is due to the violent efforts to

overcome the duodenal traction-produced obstruction, and these strong contractions may be felt by the palpating hand (they are sometimes also visible) over an abnormal area, and reveal the displacement of the stomach. The pain is not relieved by albuminous food, and milk may be badly borne. The physical and functional signs make the differentiation clear. Inflation reveals the displacement of the stomach, which is often associated with a deformed liver, a movable right kidney, and prolapse of the transverse colon. The chemical signs, in case the displacement is complicated by glandular gastritis, or by supersecretion produced by irritation of the stagnant contents, are never the same as are found in hyperchlorhydria. The water-test will exclude or reveal the myasthenia, but we would emphasize the fact that the displaced stomach is subject to the same disorders and diseases as the stomach in its normal position. The effect of a properly fitting abdominal belt and of myasthenic medication may be suggestive. But in many of the cases of gastroptosis an excessively rich gastric juice is not secreted, and there is stagnation, often fermentation, and accumulation of digestive products in the stomach. In hyperchlorhydria there is never displacement (unless the displacement be a primary or accidental association), nor motor insufficiency, nor fermentation, and during the digestive period secretion is always excessively rich, but is never abnormally large in quantity.

Hyperchlorhydria may easily be confounded with ulcer. A hemorrhage, large or small (not due to retching), would exclude the functional trouble. The subjective and the functional signs may be almost the same in the two diseases, but hydrochloric acid is not always in excess in the gastric contents of ulcer. The pain of ulcer is not relieved by taking albuminous food; it is not purely digestive, is increased by movement, may be relieved by rest in a particular position, and does not always develop in relation with the evolution of secretion and of digestion. The exquisitely painful epigastric and dorsal points of ulcer may exist even when the stomach is empty. Hyperchlorhydria is a predisposing cause of ulcer; and when doubt exists, an ulcer cure should be prescribed.

Hypersthenic gastritis resembles hyperchlorhydria even more closely than does ulcer, but the differentiation is never difficult. A large number of the cases of chronic hypersthenic gastritis, primary and secondary, are easily excluded by the associated stagnation, displacement, fermentation, or supersecretion. But a notable percentage of the cases of

primary hypersthenic gastritis, particularly in the early stage of the disease, are expressed by the objective and the functional signs of digestive secretory irritation. The gastritis is more directly traceable to dietetic errors and to alcoholism, an adequate cause for its existence being thus found. The contents, after the test-meal, contain a large quantity of mucus, cell nuclei, exfoliated and unseparated epithelium, chief and border cells and blood; and misfortune may conclusively reveal the anatomical nature of the disease in a piece of scraped-off mucous membrane. Hyperchlorhydria may be intermittent in its manifestations, and in closer relation with the state of the nervous system than with the alimentation. Some of the enumerated differential signs differ only in degree, and not in kind, and it would be impossible to say where hyperchlorhydria ends and hypersthenic gastritis begins, if it were not for the excessive mucus and the distinctive anatomical signs of gastritis.

Treatment.—Both on account of the suffering which it produces and on account of the serious diseases for which it prepares the way, hyperchlorhydria should receive careful treatment.

The two ruling principles of its treatment are sedation and the improvement of the condition of the nervous system. The irritable nervous system demands rest, which must be obtained at any cost. It may be sufficient to lighten the daily burdens, to correct excesses, or to send the patient away from home cares to lead a pleasant outdoor life. In severe cases it may be necessary to prescribe a rest-cure, for a few weeks, in bed.

Electricity, which does not relieve the pain, should be used with care; and only sedative anodal gastric or epigastric galvanization of the empty stomach should be employed. Hydrotherapy should be used to tone the nervous system, and the hot compress during digestion exerts a soothing influence on the stomach. Penzoldt recommends lavage in the evening, sometimes daily, sometimes less frequently, but we rarely employ stomach washing in this affection.

The diet is indicated by the functional signs, and should be chemically and mechanically non-irritating, leaving as little HCl uncombined as possible. The albumins of the various foods do not possess the same acid-combining equivalents, and the albumin digestive products combine more HCl as the digestive transformation proceeds. Thus, antipeptone combines, in percentage, twice as much HCl as hetero-albumose, and hetero-albumose combines double the quantity of

HCl that is combined by proto-albumose. As a result of experiments, we find that 100 gm. of the following foods, when cooked, require, at 37° C., the addition of about the following quantities of a three per thousand solution of HCl before the HCl remains free: Lean beef, 650 c.c.; veal, 710 c.c.; mutton, 630 c.c.; milk, 120 c.c.; roll, 105 c.c.; whole wheat preparations, 200 c.c.; rice, 230 c.c.; chicken, 640 c.c.; fish, 250 c.c.; cheese, 300 to 800 c.c.; lean ham, 720 c.c.; eggs, 400 c.c. The physiological action of the various foods on secretion, in addition to their acid-combining power, should guide in the selection of the diet. The diet must be largely albuminous, and must be neither physically nor chemically excitant. Milk, lean and fine-fibered fish, lean meats reduced to pulp, the soft part of small and fresh oysters, plainly cooked game, slightly cooked eggs, are all suitable articles. The cereals,—rice, wheat, oatmeal,—very thoroughly cooked and fresh, possess a high acid-combining power, and may be added to the diet list; but it should be remembered that the cereals and bread contain starch also, and the starchy foods excite the secretion of more HCl than they can combine. Vegetables and fruits must be avoided, and only enough fat in the form of fresh (unsalted) butter or cream to supply the needs of nutrition should be permitted. Spices, condiments, acids, oils, and alcoholic drinks should be absolutely prohibited. Sweets increase the quantity but diminish the acidity of the gastric juice, and in moderate quantity are beneficial in hyperchlorhydria. Milk, an alkaline mineral water, or plain water are the best drinks. Very weak tea and coffee may sometimes be permitted, but cocoa and "vigor chocolate" (Hauswaldt) are better. Dry food is very injurious, and about two glasses of fluid may be permitted with each meal. An hour's repose after each meal is obligatory. It is best, in our opinion, to permit only three meals a day.

In the treatment of hyperchlorhydria the alkalies have long held a prominent place. Bicarbonate of soda and calcined magnesia may be given during the period of free acidity in repeated doses, as recommended in the chapter on Chemical Treatment. If the pain be very severe, belladonna ($\frac{1}{50}$ to $\frac{1}{20}$ of a gr. ext.) and codein ($\frac{1}{6}$ of a gr.), or opium ($\frac{1}{10}$ of a gr. aq. ext.), may be given before each meal. Our favorite prescription is the extract of belladonna combined with two grs. of extract of coca before each meal. Opium, if it is ever advisable, should be given in full doses, as small doses of opium excite secretion and the nervous system. Cannabis indica rarely proves useful, and the bromids do no good.

We sometimes use nitrate of silver (by mouth or by means of the intragastric douche), and large doses of bismuth, employed as does Fleiner in the treatment of ulcer, are sometimes beneficial. Under the influence of this treatment the constipation and the pain may rapidly be relieved. If the constipation requires additional attention, an injection (to which a teaspoonful of glycerin may be added) should be employed, or gluten suppositories, or glycerin suppositories, or anodal sedative galvanization of the colon may be tried. Purgatives repeatedly given by the mouth destroy any good effects derived from the other remedies. The stomach must be consistently and thoroughly protected against all forms of irritation.

(B) DIGESTIVE HYPERCHYLIA GASTRICA.

Supersecretion in response to the physiological action of food is a distinct dynamic affection of the stomach. The gastric juice, as a rule, is abnormally rich in acid and ferments; but it may be normal in quality, and, exceptionally, it contains a diminished percentage of hydrochloric acid and ferments. There is no anatomical lesion of the mucosa, and the motor function is normal. Digestion is prolonged because secretion is excessive in quantity.

Etiology.—The causes of digestive hyperchylia are the same as those which produce hyperchlorhydria, and no complete explanation has yet been given of the genesis of either affection. The underlying condition may be a direct or indirect disturbance of the nerve-centers which control secretion, or excessive vital activity of the chief and border cells, or an impure blood, or a vasomotor disturbance. These patients are most frequently neurotics, or neurasthenics, or young persons guilty of excesses which, directly or indirectly, affect the brain, the central nervous system, or the abdominal sympathetic.

Clinical Description.—Digestive hyperchylia may be a latent disease, or the patient may intermittently pass a number of comfortable days. Most frequently the symptoms recur daily, sometimes after each meal, or, as the rule, only after the second and third meals of the day, the patient having an attack of eructations, belching, heartburn, pain, sometimes vomiting, and headache during the course of the afternoon and another in the evening. Vomiting and headache are more frequent than in hyperchlorhydria, the pain is often severe and spasmodic, and all the symptoms occur in relation with the

evolution of secretion and digestion. The foods which excite secretion most and remain long in the stomach produce the greatest discomfort. The appetite is usually good, and thirst is excessive. Emaciation is more frequent than in hyperchlorhydria, and the digestive and motor functions of the intestines are disturbed by the large quantity of chyme, which may be superacid and fermenting.

More characteristic than the clinical history are the physical, functional, and bacteriological signs. The abdomen is tender over the region of the stomach. Splashing and gurgling (gliding method) can be produced at a time after a meal when the normal stomach would be empty. One hour after the test-breakfast the stomach contains more than 150 c.c. of chyme; two hours after the test-meal of Sée more than 175 c.c. of contents can be expressed or estimated by the dilution and total acidity method; after the Riegel dinner more than 200 c.c. may be obtained. Consequently, the quantity of the contents is abnormally large. The specific gravity of the filtrates of the contents after the test-meals is below normal: After the test-breakfast, below 1010; after the test-meal of Sée, below 1015; and after the test-dinner of Riegel, below 1020. The ferments are present in normal quantity or in excess. There may or may not be hydrochloric superacidity, and the quantity of combined HCl is frequently diminished. The albumins are well digested, but starch digestion is decreased, Lugol's solution producing a blue or purplish color. Fehling's test for sugar is negative, or a small quantity of the copper may be reduced. There is never an abnormal quantity of gastric mucus. The evacuation of the chyme is always delayed, digestion being prolonged in proportion to the supersecretion. The prolongation of digestion is proportionately greater after Riegel's test-dinner than after the test-breakfast. When the patient eats his customary three daily meals, the stomach may or may not succeed in emptying itself between the meals. Two glasses of water are evacuated within the normal period. Secretion is inactive while the stomach is empty. There is no stagnation of solids or of liquids, but the physical properties of the contents are such as would be produced by supersecretion during the evolution of digestion. The quality of secretion may be estimated and supersecretion recognized by the emulsion-meal of Mathieu or the authors' meal for testing absorption.

The prolongation of digestion favors the development of fermentation. The most common form of fermentation in

digestive hyperchylia is acetic fermentation. The urine changes are of the same nature but more pronounced than its changes in hyperchlorhydria.

Diagnosis.—The clinical history of hyperchylia gastrica is not distinctive, but the subjective symptoms possess some diagnostic features. The symptoms all occur during the period of digestion, although the duration of this period is abnormally long. While the stomach contains no food, there are no subjective symptoms and secretion ceases. The symptoms are intensified by large meals, and by foods which remain long in the stomach and which excite free secretion. Albuminous food may temporarily diminish the pain, but the attack is prolonged by eating food. The functional signs are characteristic. The contents are too large in quantity, of abnormally low specific gravity, usually rich in acid and ferments. Digestion is prolonged without motor insufficiency, and mucus is not secreted in excess. Fermentation may or may not be present.

Differential Diagnosis.—Hyperchylia gastrica closely resembles hyperchlorhydria in many respects, and after what has already been said its differentiation from ulcer and chronic gastritis may be omitted. Digestive hyperchylia is most frequently confounded with hyperchlorhydria, myasthenia, and pyloric obstruction. The functional signs, the prolongation of digestion, the relation of the symptoms to the quantity and quality of the food, and sometimes the occurrence of fermentation distinguish it readily from hyperchlorhydria.

In both myasthenia and digestive hyperchylia the period of digestion is prolonged—in the one, on account of motor insufficiency; in the other, on account of supersecretion. In myasthenia the stomach is flabby, easily distensible, slightly retractile, and the line representing the acidity of the contents during the evolution of digestion is irregular. In digestive hyperchylia the stomach possesses its normal tonicity, retracts when it is empty, and the evolution of digestion is abnormal but regular. One and one-half hours after the administration of two glasses of water the stomach is empty in hyperchylia, but in myasthenia it contains a quantity of water proportionate to the motor insufficiency. The quantity of water which it contains may be exactly determined by introducing 100 c.c. of a one per cent. solution of sugar into the stomach, mixing it thoroughly with the contents, and subtracting 100 from the result obtained by dividing 100 by the reduced percentage of sugar in the ex-

pressed dilution. In myasthenia the delay in the evacuation of the contents is proportionate to the fluidity and the quantity of the food and to its action on the motor function. In hyperchylia the stomach empties itself most rapidly when the diet is fluid and excites little secretion. By means of our meal for testing absorption, or of the emulsion-meal of Mathieu, the portion of the total contents which is due to secretion can be estimated. This portion is abnormally large in supersecretion, and normal in quantity in myasthenia. Expression of the contents is easy in hyperchylia; in myasthenia it is always difficult and incomplete.

In the absence of a palpable tumor of the pylorus, or of the history or signs of a disease which is liable to produce obstruction of the pylorus or of the duodenum, the differentiation of hyperchylia and obstruction may require close study. The evolution and the grouping of the symptoms may be distinctive. Obstruction is persistent, obstinate, often progressive, and obstructive stagnation is a stagnation of solid and coarse food. Hyperchylia yields rapidly to appropriate treatment, and the prolonged digestion is due to the continuous dilution of the contents by supersecretion. The evolution of digestion is irregular in obstruction; but it is regular, although abnormal, in hyperchylia. The functional signs may be similar or they may be distinctive of the one or the other disease. Two glasses of water are evacuated within the normal period in both diseases, unless the obstruction is so great as to produce the severe form of stagnation; but a glass of a ten per cent. solution of sugar is evacuated much earlier in obstruction than in hyperchylia gastrica.

Treatment.—The treatment of digestive hyperchylia gastrica consists in the removal of the cause, when this can be accomplished, and in the employment of remedies to tone and quiet the nervous system and to improve the general health. Rest, open-air exercise, electricity, massage, and appropriate baths are usually beneficial. Bismuth, belladonna, and ergot are the drugs which we have found to exert a controlling influence on the supersecretion. The bismuth should be given in a single large dose (one dram) before breakfast, and the belladonna and ergot may be ordered in small doses before each meal.

But the special treatment of digestive hyperchylia gastrica is dietetic. No foods should be permitted which greatly excite secretion and which remain a long time in the stomach. Sweets should be prohibited, and the red meats increase the supersecretion. Condiments, tea, coffee, and alcoholic drinks

are injurious. The meals should be small, chiefly fluid, and separated by intervals long enough to allow the complete evacuation of the stomach. Milk, cream, cereals, eggs, calf's brain, young chicken, squab, green vegetables thoroughly cooked and passed through a sieve to insure fine division, are appropriate articles of food. The diet of hyperchlorhydria, which is chiefly albuminous, is badly borne and injurious in hyperchylia. Exclusive rectal feeding is rapidly curative, and it should always be employed for a few days in the beginning of the treatment of the severe cases.

(C) PAROXYSMAL HYPERCHYLIA GASTRICA.

Paroxysmal hyperchylia gastrica—gastroxynsis (Rossbach), gastroxia (Lépine), intermittent or periodical gastrosuccorrhea—is a dynamic affection of the stomach characterized by severe headache, by vasomotor disturbances, and by the supersecretion of an excessively acid gastric juice, rich in ferments; it recurs in paroxysms, separated by intervals of perfect health. It is a cerebrogastric trouble, due to the reaction of an irritable and exhausted brain on the solar plexus (Leven); or to excessive secretion excited by an irritable cortex, the wide-spread vasomotor disturbances proceeding from the gastric mucous membrane (Rossbach); or it is due to a primary disturbance of the vasomotor center in the medulla, of which the gastric and cerebral symptoms are expressions (Rosenthal). The affection is very closely related to migraine, of which it is probably a form.

Etiology.—Gastroxynsis is a disease of school-children, of students, and of brain-workers, and appears to be almost exclusively met with in the male sex. The attacks occur periodically, being excited by mental overwork. Abuse of tobacco is said to be another cause. The disease is sometimes met with in persons who can not be accused of mental overwork, and it may be excited by intestinal auto-intoxication and by biliary and renal colic. It occurs as a symptom of sclerotic bulbar lesions.

Clinical Description.—The attacks begin suddenly, without prodromal symptoms, unexpectedly, either during digestion or during the period of gastric repose, but most frequently during the second half of the night, with severe, colicky gastric pains which may or may not be accompanied by headache. The pain may radiate over the abdomen and into the back and shoulders, and be so severe as to produce collapse. Vomiting

soon follows, but it affords only partial and temporary relief, and recurs again and again after short intervals. The vomit consists of the accidental contents of the stomach and of a gastric juice rich in hydrochloric acid and ferments and tinged green with bile. If food be in the stomach when the attack begins, the vomit is strongly acid, the acidity being due to an excess of free and a large quantity of combined hydrochloric acid. After the stomach is emptied secretion continues; and the vomit consists of a greenish fluid of a total acidity of 20 to 40 and a specific gravity of about 1006, and containing the ferments of the stomach. The acidity is due almost entirely to acid phosphates (6 to 12) and to free hydrochloric acid. There is no noteworthy increase of gastric mucus and no blood, but a few cell nuclei are present. The quantity of fluid vomited is largely in excess of what has been swallowed. Secretion is active and continuous. Headache may be the predominant symptom, and it is sometimes agonizing, or it may be but slight. Appetite is lost and the patient complains of great thirst. During the interval between the attacks the well-nourished patient experiences no discomfort during digestion, the appetite is good, and the functional signs are normal.

Diagnosis.—Paroxysmal hyperchylia gastrica is characterized by intermittent painful attacks of supersecretion and vomiting, separated by intervals of normal gastric digestion. Locomotor ataxia and multiple sclerosis may be accompanied by similar paroxysms, and in every case the signs of these diseases should be sought. Paroxysmal supersecretion may be the initial symptom of tabes dorsalis.

Treatment.—The patient should be put to bed in a darkened room, and hot water administered. If given in the beginning of the attack, the hot water may abort it, and later it will excite profuse vomiting and afford relief (Rossbach). Phenacetin, antipyrin, or antifebrin may also be administered. Caffein may give relief if administered early in the attack. The Winternitz compress is beneficial. The best treatment in the beginning of the attack is a thorough stomach washing, followed by a purgative dose of calomel, and by morphin and atropin hypodermically. During the attack food should not be given by mouth, but the patient should be nourished exclusively by enemata.

The preventive treatment consists in the avoidance of mental fatigue and of intestinal stagnation and decomposition, in digestive hygiene, and in attention to the general health. Errors of refraction should be corrected by glasses.

2. ADENASTHENIA GASTRICA.

A dynamic affection of the stomach, characterized by a diminished and a poor secretion without an anatomical lesion of the mucous membrane, is known as adenasthenia gastrica (subacidity, anacidity, hypochlorhydria, hypochylia).

Etiology.—The disease is less frequent than adenohypersthenia gastrica, and may be met with as a particular form of neurasthenia gastrica and in hysteria and the psychoses. But the trouble may develop in those who are neither neuropathic nor neurasthenic, and may be a consequence of railway shock, of fright, of sorrow, of depressing moral influences, or of chronic subnutrition. It is sometimes found in the severe anemias and in chlorosis, and is a symptom of many of the acute febrile diseases. Adenasthenia gastrica predisposes to intestinal diseases.

Clinical Description.—Gastric subjective symptoms may be entirely wanting or may consist in slight digestive discomfort. The appetite is often poor, but if the intestines are healthy and enough food is taken the general nutrition is well maintained. The urine is often highly acid, the diminution of its acidity during digestion does not occur, and uric acid frequently precipitates after the urine stands for a few hours. There may or may not be diarrhea, which is as frequent in adenasthenia as constipation is in adenohypersthenia gastrica.

The patient feels unfitted for work, and no adequate cause can be found for the mental depression. It is usually difficult to persuade the adenasthenic that there is nothing serious the matter, the thoughts and feelings, it would seem, taking all their color from the depressed function of the stomach.

The functional signs are the distinctive characteristics of the disease. The filtrate of the contents obtained after the test-breakfast contains no free HCl (H), the albuminoid affinities for acid are not satisfied, and in some cases the combined hydrochloric acid (C) is present in mere traces or is altogether wanting. The ferments may be present in proportionately greater quantity, but, as a rule, the pepsin is diminished in proportion to the diminution of the total HCl, but not of the free HCl alone. If hydrochloric acid be administered ten minutes before the expression of the test-breakfast contents, both tube peptonization and milk curdling are sometimes as active as in health, for the mother substances of the ferments are present in greater quantity than are the converted ferments. Another characteristic is the influence

of electricity. Cathodal dorsogastric galvanization for ten minutes, with a current of five to ten milliamperes and a current density of $\frac{1}{20}$, during the second half hour of the digestion of the test-breakfast, improves, or even removes, the secretory depression. In the gastric contents after the test-breakfast, or after the test-meal of Germain Sée, there is only a moderate quantity of syntonin and propeptones, a large quantity of the albumin remaining undissolved. The acidulated tube pepsin tests give a digestive percentage less than normal, and the dilution is always less active than the undiluted test. There is no motor insufficiency in spite of the absence of the motor stimulation exerted by hydrochloric acid, the test contents being less liquid than normal. There is little mucus, no excess of formed elements, and no fermentation, except, irregularly, there may be a strong odor of butyric acid. Starch digestion is greater than in the normal stomach. The specific gravity of the contents is abnormally high, and the total quantity of secretion is frequently diminished.

Differential Diagnosis.—The functional signs, the genesis, the vague subjective symptoms, and the absence of a discoverable disease of any organ are the salient features of adenasthenia. It may be confounded with carcinoma, with asthenic gastritis, and with atrophy of the gastric glands.

In carcinoma the secretory signs may be similar, but here the resemblance ends. The disgust for certain foods, the relation of the gastric symptoms to the quality of the food, the progressive character in spite of the purposive treatment, the bacteriological signs, possibly the physical signs of a tumor, the motor insufficiency, gastric pain, excess of mucus, etc., do not exist in adenasthenia. Confusion is not likely to occur unless an attempt be made to base the diagnosis on the narrow and misleading hydrochloric subacidity alone.

The differentiation of the functional disorder and asthenic gastritis is not difficult. In asthenic gastritis there is always an excess of mucus, and the secretion can not be excited so readily by stimulants and by electricity. The subjective symptoms of gastritis are in relation with the physical qualities of the food. The causation and the mode of beginning may suggest the nature of the trouble. In gastritis there may be symptoms, such as nausea and vomiting, which are not found in adenasthenia. In asthenic gastritis the ferments are persistently diminished, which may be the case in adenasthenia. The dynamic affection is very rare.

Treatment.—The treatment is excitant, the aim being to restore the secretory power and to tone the nervous system.

The first object is to reduce the life of the patient to a physiological basis and to restore tone and vigor to the whole system by the use of tonics and of hygienic remedies suited to each individual case.

As a chemical remedy, hydrochloric acid with pepsin may be given in two or three doses during the period of digestion, but with no hope of producing directly more than a purely chemical action. The effect of the acid on intestinal digestion should be watched. Peptones may, however, be administered before meals to excite secretion, acting in this respect as physiological remedies.

The local treatment is physiological and excitant—the Scottish douche, electricity, the intragastric chlorid of sodium douche, in the manner recommended in the chapter on General Medication.

The diet should not vary much from that of health—meats in moderate quantity and finely-divided cereals (since the digestion must be done by the intestines), vegetables, sweets, and fats. The starchy foods are well digested and they should be particularly recommended. Sweets not only are useful as food, but as a physiological means of exciting secretion. Beer, wines, ale, cider, tea, and coffee may be recommended in moderation. Fine physical division of all of the food is demanded by the intestines, on which the brunt of digestion falls. The food to be taken during the twenty-four hours should be divided into three or four equal portions.

Calumba, gentian, cinchona, and nux vomica may be combined with aromatic tinctures, and administered half an hour before meals. The effect of the excitant treatment should be carefully watched and not pushed too vigorously, and should be stopped as soon as the secretory activity is restored. Three grains of orexinum basicum may be given in a gelatin capsule daily, in the morning at 10 o'clock, with a cup of bouillon, but the remedy should not be continued longer than five days without an intermission. A powder composed of common salt and chlorid of calcium, taken in a wineglassful of a mildly alkaline water half an hour before meals, is an excellent stimulant of secretion.

CHAPTER III.

THE MOTOR DYNAMIC AFFECTIONS.

I. SPASM OF THE CARDIA.

On the introduction of the stomach-tube a slight resistance is felt by the experienced finger just before the entrance of the tube into the stomach. This spasmodic contraction of the cardia is physiological, and is often manifest to a person when a large or somewhat irritating bolus is swallowed. It is on account of this spasm that swallowed corrosive poisons are arrested, and are so energetic in their action on the lower portion of the esophagus. Even a full swallow of water occupies about twelve seconds in passing through this normal point of constriction, as indicated by the interval separating the two deglutition sounds.

Pathologically, the normal constriction or contraction of the cardia may be increased both when the cardia is and is not the site of organic disease. In esophagitis, ulcer of the cardia, and sometimes in carcinoma, the stenosis of the cardia is partly spasmodic. Spasm of the cardia occurs also as a dynamic affection without a local anatomical lesion. The stricture of the cardia is in such cases characterized by the ordinary signs of simple spasm, in association with the stagnation or retention of the swallowed food and the secretions above it, and with the accumulation of gases in the stomach below it.

Etiology.—Cardiospasm (sometimes called esophagismus) is not a frequent disease. It may occur at any age, and is most common in the neurotic and the arthritic. Cerebral excitement is sometimes the occasion of its generation, but the origin of the reflex spasm may be in any of the organs closely connected with the nerves of the cardia. Frequently the abuse of tobacco is the most active factor in its causation. It is sometimes produced by hyperesthesia of the cardia, which may be engendered by very hot or very cold drinks, and by the abuse of condiments. Spasm of the cardia sometimes results, by reflex action, from the habitual swallowing of air, and we have seen two cases developing as a result of arteriosclerosis, there being at the same time erosions of the gastric mucosa.

Clinical Description and Objective Signs.—Spasm of the cardia may be acute, paroxysmal, or recurrent, or it may be chronic. The acute attacks last only a few days, and are characterized by dysphagia, by absence or delay of the swallowing sounds, and by strong, but eventually yielding, resistance to the introduction of the large and moderately stiff stomach-tubes. The food accumulates in the esophagus, and after a few mouthsful an effort to force it into the stomach becomes necessary to relieve the pressure behind the sternum. The cardia, however, does not always yield, and the food may be regurgitated into the mouth, and attacks of dyspnea, resembling so-called asthma dyspepticum, may occur. In the chronic form, which is fortunately rare, the dysphagia and other symptoms occur after each meal, but its long course, running often for months and years, may be interrupted by periods of marked improvement.

Clinically, three degrees of the chronic affection may be distinguished, two being characterized by esophageal stagnation and the other by esophageal retention of the swallowed food. The form with food retention occurs, with few exceptions, only where the lower end of the esophagus is dilated; the tube introduced in the morning before breakfast withdraws remnants of food eaten the previous day.

In the stagnation form the esophagus is either found empty in the early morning or it contains a noteworthy quantity of mucus and saliva. The stagnation form may be mild or severe. In the mild form the spasm occurs chiefly during the meal; in the severe form the spasm is more persistent and obstinate. In the mild form the entrance of food into the stomach is delayed and difficult, but is effected before the next meal; in the severe form the food is forced into the stomach only after special efforts and devices, and the swallowed secretions and the secretion of the esophagus accumulate between meals, and particularly at night. In the mild form the patient feels the food stop before entering the stomach, but after a few more mouthsful or a few moments' delay the spasm yields and the food enters the stomach, to the relief of the patient. In the severe form the mere delay is not sufficient, but the accumulated food must be forced through the cardia, often after drinking a glass of water. A deep inspiration is taken, and an expiratory effort is made or the thorax is compressed by the hands, while the glottis is kept closed, sometimes during repeated acts of deglutition. In the mild form the trouble ends with the swallowing of the

meal. In the severe form the accumulation of the secretions may manifest itself by non-alimentary esophageal vomiting.

In the retention form the spasm can not be completely overcome, either involuntarily or by voluntary effort, and more or less of the swallowed food remains in the esophagus, which is usually dilated. The dilatation is nearly always symmetrical, though it may be unilateral, irregular, or sacculated. Retention produces much more serious symptoms than simple stagnation, such as dyspnea, palpitation, fermentation, putrefaction, and esophageal alimentary vomiting. The dyspnea occurs not only during the meals but also after moderate exercise or effort, or it may be paroxysmal and nocturnal. The palpitation occurs intermittently in relation with the filling of the dilated pouch, and is rapidly relieved by its evacuation. The fermentation is usually butyric or lactic, and putrefaction is rare. The vomiting often occurs without effort or warning, and may take place during sleep. The unfortunate patient sometimes awakens with dyspnea, cyanosis, and palpitation, and with the mouth already full of the regurgitated material.

In all forms of the affection the cardia may show favoritism in permitting the passage of the various sorts of food: now hot, now cold, now solid, now fluid food being arrested. The patient must stop eating once, twice, or several times during the meal to allow the cardia to relax, or he must force the food which has accumulated in the esophagus through the resistant cardia into the stomach. In the mild stagnation form it is only necessary, as a rule, to wait for a few minutes, but in the severe stagnation and in the retention varieties of the affection active and voluntary efforts must be employed, and the frequency of these forcing efforts is proportionate to their inefficiency and to the smallness of the capacity of the esophagus. If the esophagus is largely dilated but empty when the meal is begun, the patient may be able to eat an ordinary meal before forcing the food into the stomach. The patient, after the meal, commonly retires to a private corner, takes a glass of water, inspires deeply, closes the glottis, contracts the thorax by means of the expiratory muscles, or by compressing with the hands, and repeats the procedure until the contents of the esophagus are completely or in part forced onward into the stomach or backward into the mouth. Patients often feel water pass the lump of retained food and hear it gurgle into the stomach. The spasm may be excited in some cases only by the act of

swallowing, the sound or tube passing into the stomach without resistance.

In addition to the esophageal stagnation and retention, the spasm of the cardia may often cause very distressing tympanitic distention of the stomach. The distention may be produced by swallowed air or by the gases of chemical decomposition or of fermentation in the stomach, the cardia again closing after being forced by the swallowed food, or contracting as a result of the irritation of the mucous membrane of the stomach. This reflex spasm of the cardia may exist without dysphagia. The distention of the stomach produces discomfort, dyspnea, palpitation, and sometimes painful gastrospasm, the attacks lasting from a few minutes to several hours, the pylorus being also simultaneously closed.

Although the clinical expression of spasm of the cardia is quite well defined, the exploration with the sound furnishes the most exact information. The very soft, flexible tube passes in easily for about 14 inches, removing the contents of the esophagus, but it is rarely possible to force it through the spasmodic stricture. With the tube in this position, a glass of water can be introduced into the esophagus and withdrawn by siphonage before it enters the stomach, particularly where the esophagus is dilated. The stiff English or German stomach-tubes can usually be introduced into the stomach after gentle pressure is patiently employed for one or two minutes, but the flexible English esophageal sounds of large caliber pass easily into the stomach. Sometimes a sound or stiff tube may be introduced without a perceptible resistance at the cardia, the spasm being excited only by acts of swallowing. In the majority of the cases of esophagismus gagging is produced by the use of the tube, and the patient often gives a history of spasm in other parts than the cardia. The effect of bromids, combined with atropin hypodermically, is so marked in the relief of spasm of the cardia that the ease with which the tube can be introduced while the patient is under their influence is of value in the differential diagnosis. If the tube introduced before the evening meal removes no food from the esophagus, there is the mild stagnation form. If food be removed at this time, and if the esophagus is empty in the morning before breakfast or contains only mucus and saliva, the severe stagnation form exists. If the esophagus contains food in the early morning before breakfast there is esophageal retention, and if a noteworthy quantity of contents is removed the esophagus is also dilated. The dilated esophagus may hold a pint or more. When the

esophagus is not empty in the early morning it is our custom to wash out the esophagus and then to introduce a glass of milk, after first pushing the tube well into the stomach. The tube is next withdrawn and the patient is given a glass of water and directed to try to force the water into the stomach. Ten or fifteen minutes later the tube is introduced, and the degree of obstruction of the cardia, and of dilatation of the esophagus, is proportionate to the quantity of water which is recovered. The tube may then be pushed on into the stomach, and the obvious inferences may be made from the withdrawal of milk. Before the entrance of the tube into the stomach it is not possible to inflate the stomach by pumping in air.

The deglutition sounds are either modified or absent. The first sound is usually delayed, and the second sound, if heard at all, is deferred several minutes or longer. Swallowing is followed by complete silence if nothing enters the stomach, and the deglutition sounds may be heard only after the patient voluntarily presses the arrested food or fluid through the cardia.

Differential Diagnosis.—Obstruction of the cardia may be organic or spasmodic. The non-spasmodic obstruction may be due to compression by tumors of the mediastinum, to Pott's disease, or to neoplasms arising from the vertebral column, or to aortic aneurysm. The symptoms incident to these diseases should be sought for in the obscure cases. Organic strictures may be produced by cancer, ulcer, sacculation, the cicatrization after burns or after the swallowing of corrosive poisons, and varicose esophageal veins.

There are signs which distinguish organic from spasmodic stenosis. Organic stricture is persistent: spasmodic stenosis may be intermittent. Atropin and the bromids are without noteworthy influence on organic stricture, but often relax the spasm. Stiff sounds pass a spasmodic stricture more readily than the soft tubes, while the reverse is true of organic stricture. But spasm may coexist with organic diseases of the cardia, and by the disappearance of inflammatory swelling the spasm may be diminished; improvement is, consequently, no distinctive criterion. The differentiation should be based on the group of symptoms or signs.

Carcinoma is most frequent between thirty and fifty, and is regularly progressive. Hemorrhage is not rare, and the blood is unlike that which has been blackened or browned by the acid of the gastric juice. Blood, however, may be vomited after its entrance into the stomach. The cardia is involved in eight

per cent. of all cancers of the stomach, but the neoplasm almost invariably extends to or originates in the stomach or esophagus; consequently, the functional and bacteriological signs of cancer of the stomach are usually present, and are valuable aids in making a decision. The esophageal contents are usually offensive, and blood and particles of the neoplasm may be removed with the tube. Soft tubes enter the stomach more readily than stiff sounds, and the caliber of the stricture may be rapidly enlarged by ulceration, or diminished by swelling and by the growth of the tumor. Cancer is the most frequent disease of the cardia, and the emaciation and loss of strength are greater than should be produced by the patient's diet.

Ulcer involving the cardia is most common in men who have passed the fortieth year. It is manifested by nausea, vomiting, and a raw, tearing pain excited immediately by swallowing, and located behind the sternum near the left sixth intercostal space; from this point the pain may radiate into the epigastrium, back, and shoulders. Hemorrhage is frequent, and the introduction of the sound is very painful, always injurious, and often impossible. Cicatricial stenosis, persistent or progressive, is more readily passed by small sounds, and is preceded by the signs and symptoms of destructive inflammation. The gastric juice in ulcer of the cardia is usually excessively acid, and many of the signs and symptoms of gastric ulcer accompany it.

Sacculation of the lower end of the esophagus is exceedingly rare, this trouble being located usually at its upper extremity or in its central third. The esophageal contents often contain pus and sometimes blood, and often ferment, or sometimes putrefy. The fermentation is usually butyric or lactic. Both the tube and the sound pass readily when the sac is empty, but, as a rule, only then. No abnormality of the functions of the stomach is produced by sacculation.

Varicose esophageal veins are accompanied by cirrhosis of the liver, or by other causes and signs of obstruction of the portal circulation.

Spasm of the cardia, rarer than either cancer or ulcer, occurs at all ages, but most frequently in neurotic and arthritic patients. The course is long, intermittent, or remittent. The spasm is palpably relieved by antispasmodics; stiff sounds pass easier than the soft tubes, and no change in the functions of the stomach takes place. Consequently, normal functional and bacteriological signs are against ulcer and

cancer. There is no spontaneous bleeding, nor blood in the opening of the tube. The other signs and symptoms of spasm of the cardia have been enumerated in its clinical description.

Treatment.—The valuable remedies in the treatment of spasm of the cardia are few. The etiological and constitutional treatment should not be neglected, and electricity may be tried. Cervico-esophageal sedative polar galvanization is the preferred form, but it is no more and no less valuable than intragastric anodal galvanization.

The use of the esophageal sound is in itself sometimes curative, and is the mainstay of any plan of treatment. A large flexible sound should be passed through the cardia and left in place for several minutes. The sound should be used once a day in the stagnation form, preferably before breakfast. If esophageal retention is present, the sound should be introduced before each meal. The lower end of the tube may be smeared with an ointment of cocain (Rosenheim), or a small piece of sponge attached to a silk thread running through the tube may be placed within the eye of the tube, saturated with a solution of cocain, which is squeezed out after the tube is against the cardia by introducing a tube-guard (Penzoldt). If the esophagus is dilated, its contents should be washed out at bedtime. It is a most excellent plan to introduce all the food through the stomach-tube.

Bromid of potassium is often palliative, but sometimes does little good unless given in large doses. The spasm often returns after the bromism subsides, but the remedy is indicated as a palliative, unless there is excessive hydrochloric acidity of the gastric contents. Nitrate of silver is then much better, and in the simple cases also it often proves of more service than do the bromids. The extracts of coca and of hyoscyamus washed down with chloroform water are very beneficial. These remedies should be given half an hour before meals. It is very important to control excessive hydrochloric secretion and to prevent butyric acid fermentation both in the esophagus and stomach.

The diet is often too restricted. The one essential is that it should not be irritating, and condiments and acids and half-mastication of the food should not be permitted. All the food and water should be taken in three meals, nothing being permitted during the intervals. The diet should also satisfy any peculiarity of the spasm, which sometimes shows a repugnance for certain foods. In other respects, the

diet is selected in reference to the condition of the stomach and the intestines, the needs of nutrition, and also to the condition of other organs when diseased, and coarse or solid food may sometimes be employed as a means of dilating the cardia.

II. SPASM OF THE PYLORUS.

The pylorus is physiologically the most important sphincter of the digestive tube, exceeding in the variety and value of its work not only the cardia and the anus, but also the ileocecal valve and the duodenojejunal constriction. It controls gastric digestion by regulating the time during which the food is subjected to the churning movements, to the transforming gastric jnice, to the absorbent surface of the stomach, and to the action of the saliva. It regulates the supply of nutritive material to the intestines without the interference of the will. It separates the two chief divisions of the digestive tube, protecting both the stomach and the intestines. Normally, there is no reflux through it to disturb the stomach, and no harm should come through it to the intestines. It controls the gateway to nutrition. But its work is not done without favoritism, for it often protects the intestines at the cost of slow starvation and of injury to the stomach. While acting normally it may not do harm, and the disturbance produced by it when diseased corresponds to the importance of its work in health. One of its chief functional disorders is spasm.

Spasm of the pylorus is either primary or secondary. The secondary spasm of the pylorus is common in ulcer, in cancer, in hypersthenic gastritis, in adenohypersthenia gastrica, and in excessive secretory activity accompanying stagnation and retention. It consequently plays an important part in the evolution and in the genesis of the symptoms of acute and chronic hypersthenic gastritis, and of complicated forms of myasthenia. The disease produces the spasm, and the spasm causes or increases stagnation or retention, with consequent irritation of the mucous membrane of the stomach, excessive peristalsis or gastrospasm, and often vomiting. A vicious gastric circle thus becomes established.

But spasm of the pylorus occurs frequently as a primary dynamic affection—a morbid entity with a distinctive expression and with a proper rational treatment. Its existence can be established only by clinical observation, and its frequency by the recognition of the vicious circle of which it forms a

part, and by observing the method by which this same circle is broken.

Etiology.—Like spasm of the cardia, spasm of the pylorus is most common in neurotic and arthritic patients, and it may be occasioned by shock, anxiety, worry, or prolonged mental or moral strain. It may occur as an accident, the mere manifestation of a dietetic error, or the effect of very cold drinks, the protection of the intestines necessitating an acute disturbance of digestion. Spasm of the pylorus is an almost constant accompaniment of gall-stone colic and of pyloric hyperesthesia, and it may produce complete occlusion for several days. It is common in the acute diseases of the intestines, particularly when nausea and vomiting are present. When the body of any of the hollow organs that are closed by sphincters becomes relatively weak, the antagonistic sphincter muscle may remain contracted. This is very likely the genesis of pyloric spasm occurring in old age, and during convalescence from severe diseases.

Clinical Description.—Pyloric spasm, like spasm of the cardia, is digestive and periodical, or it is more persistent. Consequently, it produces either stagnation or retention and their respective consequences.

One of the most common symptoms is gastric flatulency. The swallowed air and the gases of chemical decomposition and of fermentation when it is present, accumulate in the stomach, and are got rid of by belching or finally by the relaxation of the pylorus and their rapid escape into the duodenum. This sudden relief by the rapid and perceptible evacuation of the stomach is characteristic.

In the severe cases there are often painful gastric peristalsis and agonizing pyloric colic, which may end suddenly with the relaxation of the pylorus, or may recur intermittently until the stomach is empty, or cease after copious alimentary vomiting.

There is no discomfort when the stomach is empty, but the pain may continue as long as the food and digestive or fermentative products remain in the stomach. In retention the symptoms may become continuous and the stomach may ultimately reject everything introduced into it.

The objective signs are much more characteristic than the subjective symptoms. In some cases the pylorus can be felt as a firm cylinder moving up and down with the diaphragm. During digestion there is no intermittent palpable bubbling through it, nor does it become alternately hard and soft. The intermittent pyloric spurt is not heard either after

a glass of water or during the digestion of a meal. The pyloric evacuation sound may be absent when there is visible or distinctly palpable gastric peristalsis. These abnormalities disappear, either spontaneously or intermittently, under the influence of antispasmodic treatment.

Artificial inflation of the stomach is easy and the viscus remains distended much longer than when it is normal. The gas or air can not be massaged into the duodenum as in health.

In the simple cases gastric absorption is normal. Secretion may be normal or may be excessive, but the abnormality usually disappears with the restoration of the motor function. The characteristic functional sign is intermittent stagnation or retention. After the test-breakfast there is an excessive quantity of contents of high specific gravity (above 1015) and containing an excessive quantity of digestive products. There may be mild or severe stagnation or retention, but the motor insufficiency in a particular case, whatever be its degree, may spontaneously and suddenly disappear, or may be rapidly improved by sedative and soothing medication. This distinctive sign is never met with in myasthenia. If stagnation, as a result of pyloric spasm, occur in adeno-hypersthenia gastrica, it is rapidly relieved by a milk diet with large doses of the alkalies, but the excessive secretion is more rebellious. In stagnation or retention due to myasthenia the excessive secretion may be controlled, but the motor insufficiency disappears very slowly and gradually.

Treatment.—The treatment of primary spasm of the pylorus is very simple, but not always rapidly successful. Measures to improve the tone and strength and to allay the irritability of the nervous system are naturally in place. If retention be present, the stomach should be daily washed out, but in stagnation only when there is fermentation.

The diet should be soothing, mild in its action on secretion, and easily evacuated, and should also be selected with a view to its resistance to fermentation if the motor insufficiency be pronounced. Consequently no particular diet will suit every case.

Of the physical remedies, cervicogastric galvanization and the Winternitz or a hot compress (coil over moist flannel) may be employed during digestion. Nitrate of silver is valuable if the stomach is morbidly sensitive or secretes excessively, or when there is hyperesthesia of the pyloric mucous membrane. The extracts of coca and belladonna should be given before each meal, and if there is much pain codein phosphate should be given hypodermically, or chloral hydrate

may be given by rectum. It is bad practice to give these remedies by mouth for the relief of painful pyloric spasm. Hot drinks may be given and heat may be applied externally, or the stomach may be washed out and left empty.

III. GASTROSPASM.

Tonic spasm of the stomach may be a symptom or, rarely, a distinct morbid entity occurring without any organic change in the mucous membrane.

Etiology.—Symptomatic gastrospasm occurs in pyloric obstruction, at times during the digestive period, and at times during the period of normal repose, on account of the effort of the stomach to evacuate the retained chyme. But the stomach when empty again relaxes. In hypersthenic gastritis and in adenohypersthenia gastrica the stomach may be resistant and rigid, and also small when it is not full. The spasm of the stomach in these diseases and in ulcer, like pyloric spasm, is due to the irritation or hyperesthesia of the mucous membrane. The chronic asthenic gastritis which occurs in advanced arteriosclerosis may be accompanied by paroxysms of painful gastrospasm, and these attacks may occur either when the stomach is empty or during digestion. Tabes may rarely be manifested by crises of gastro-intestinal tonic spasm. A small, contracted, hypertrophied stomach in a permanent form is a sequel of long rumination. In cases of chronic and periodical vomiting, with complete gastric intolerance, the stomach is contractured, and the same condition is a result of acute nicotin-poisoning and probably occurs in meningitis.

Clinical Description.—Primary gastrospasm may or may not be painful, or may be only periodically painful, and particularly so after the ingestion of food. The sensation of constriction of the stomach, often felt and complained of by the patient, is not always relieved by emptying the stomach. The capacity of the stomach, and consequently its surface area also, is small. If there be vomiting or discomfort, or even severe pain, a quantity of food that is large as regards the size of the stomach always produces these symptoms, but small meals may be well borne. Inflation of the stomach excites nervousness, local distress, and pain. When the stomach is empty the epigastrium is depressed, and may become prominent after meals. Above the depression, and well up under the left costal border, the rigid contractured

stomach can sometimes be felt moving up and down with the diaphragm. The epigastric prominence is produced by the hard, smooth stomach, manifesting no palpable peristaltic movements. In neither case is the stomach tender. There is no chemical abnormality of the test-breakfast contents if the bread has been thoroughly masticated. The stomach may be too rapidly evacuated, but more frequently the pylorus is also tightly closed.

Diagnosis.—The diagnosis of the dynamic affection—the course of which may be long or short, remittent or intermittent, beginning and ending suddenly without apparent cause—must be made by exclusion and by the presence of its physical signs. The gastrospasm can only be rightfully considered primary in the absence of the diseases of which it may be a symptom. Fortunately, the signs of these diseases are distinctive.

Treatment.—The treatment is almost exclusively dietetic. The bromids only act as palliatives, cannabis indica is uncertain, and belladonna is of no benefit. Aconitia and codein are of the greatest value, and in combination have a marked influence on the trouble. Sedative galvanization and a hot compress may be tried. Vigorous massage is also beneficial.

The diet in the beginning should be soothing and small in quantity, and when well borne nothing is better than hot milk. The quantity of the milk should be gradually increased, and after a week cereals, and, later, meats, should be added to the diet, the object being gradually to render the stomach tolerant of larger and larger quantities of food which excites it little and leaves it rapidly.

IV. TORMINA VENTRICULI NERVOSA.

Excessive and visible peristalsis of the stomach as a simple dynamic affection is very rare. The phenomenon is nearly always a symptom either of pyloric or duodenal obstruction or of gastroptosis.

Peristalsis may infrequently be palpable or visible during normal digestion. Pathologically, gastric peristalsis may also occur periodically, or persistently during the period of normal repose. It is then either a symptom, the stomach containing either food or gas, or possibly it is a distinct morbid entity.

The dynamic form occurs in neurotics and neuropaths—particularly in hysteria and in neurasthenia. It may then

exist alone, or may be associated with excessive intestinal peristalsis.

The visible and palpable peristalsis may occur when the stomach contains no food, but it is most active during digestion. It may also be excited by gently stimulating the skin over the epigastrium with the tips of the fingers, or by cold, or, better, by introducing food or cold water into the stomach. It seems, at times, to be a mere effort to rid the stomach of gas, and often ceases with its evacuation. Excitement may either stop or start it.

As the patient lies quietly on the back, the peristaltic wave emerges from beneath the left costal border, rises prominently into view, and falls beneath the linea alba, to rise again slightly, and finally to disappear at the pylorus. The wave may also be antiperistaltic, and the circuit may be traversed several times in a minute. The agitation may be accompanied by churning and gurgling noises, but the peristalsis is never painful. It may continue day and night, and produce insomnia. Seldom nausea and vomiting and belching occur. There are no secretory or bacteriological signs. The patient complains of the perceptible movements in the abdomen and of the peculiar uneasiness which accompanies them.

The **diagnosis** is readily made by inspection and palpation, and it is not difficult to locate the trouble in the stomach by the aid of the physical signs. The affection may exist without the peristaltic waves being visible, the subjective sensations described by the patient first directing attention to it. The dynamic affection occurs in nervous persons in the complete absence of signs of obstruction to the evacuation of the stomach and in the absence of gastroptosis, and is relieved by codein, electricity, and rest. On careful study, the simple dynamic affection will be found exceedingly rare.

The **treatment** consists of rest in bed, an indifferent diet, aud strong intraventricular or epigastric faradization, or, preferably, anodal sedative galvanization. The general nervous system should be given tone and strength by hydrotherapy, good hygiene, and reconstituent medication.

V. ERUCTATIO NERVOSA.

Belching is common both in health and disease, and occurs or is voluntarily induced in order to relieve the stomach of accumulated gas which has been swallowed with the food or drinks or saliva, or which has been generated in the organ

chemically or by germs, or which has been regurgitated from the intestines. This simple or symptomatic belching ends with a few easy eructations of the gas contained in the stomach.

Eructatio nervosa is a dynamic affection of the stomach characterized by periodical and paroxysmal attacks of rapidly repeated and often very noisy belching. It is essentially a reflex effort, aided sometimes by volition, to relieve a dominant and peculiar sensation. The affection is composed of two factors—the sensation associated in consciousness with the accumulation of gas in the stomach, and the effort to relieve it by belching. The stomach actually may or may not contain an excess of gas.

It is claimed by some that, in this affection, the stomach draws in and drives out forcibly atmospheric air, after the manner of a Politzer inflator, the suction and expulsion being produced by relaxation and contraction of the muscular layer. Oser notes its occurrence in individuals who manifest in other ways excessive peristaltic activity, and maintains that the cardia is not relaxed, as very great increase of abdominal pressure does not expel the gas. In Stiller's opinion, paresis of the cardia is the essential condition, but aided by some expulsive force, it being impossible to exclude contractions of the stomach. He notes, in support of this theory, that the affection is often associated with paresis or spasm of the throat, esophagus, or stomach. Both Oser and Stiller consider eructatio nervosa distinct from esophageal belching.

Bouveret thinks that the essential factor is clonic spasm of the pharynx, and is more than inclined to consider the affection a neurosis of the pharynx, or aërophagia. As a result of close observation, he separates the belching into two parts—the swallowing and the expulsion of air. The convulsive swallowing is accompanied by tight closure of the lips and mouth, by elevation of the larynx, and by a single short but audible sound. The expulsion is produced by the contractions of the esophagus, and is accompanied by a long, loud, vibratory, and characteristic noise. Some of the swallowed air may enter and accumulate in the stomach and aid in the production of the second sound by its occasional expulsion.

None of these explanations is satisfactory, and least of all the one which makes the affection a simple aërophagia. A part of the seemingly hysterical effort does not constitute the disease, which is the reflex and sometimes partly voluntary and repeated employment of the usual means of getting gas out of

the stomach, and in eructatio nervosa, also in order to relieve a peculiar and uncomfortable gastric sensation. It is the sensation that bothers the patient more than does the belching, and that distinguishes the affection from the nervous and usually hysterical swallowing and noisy expulsion of air from the esophagus. Aërophagia nervosa is an imitative affection, a play to an audience or a plea for sympathy—a simple psychosis. The attacks are immediately arrested by holding the mouth open, thus rendering the swallowing of the air impossible.

If a tube be introduced into the esophagus, air is drawn in during each inspiration. The same phenomenon is noticeable if, by muscular action or relaxation, either end of the esophagus becomes open during respiration. In voluntary esophageal belching the mechanism is very simple. During an inspiratory act while the glottis is closed, the larynx is lifted upward and forward, and the air rushes in, sometimes with an audible sound. The larynx is allowed to fall back, and expiratory effort while the glottis is closed forces the air out with more or less noise. In aërophagia nervosa the procedure is the same, but is involuntary, and the contractions of the esophagus chiefly, or unaided by expiration, force out the air. If the cardia be open, the opening of the pharyngeal end of the esophagus by lifting the larynx upward and forward enables the gas to escape, provided (as is nearly always the case when the stomach is distended with gas or contracted on its contents) that it is subjected to a sufficient pressure to overcome the resistance of the atmosphere. If the cardia is not open it may yield to the slight aspiratory suction exerted during the expulsion of the esophageal air by the full current of a forced expiration, or to the suction exerted by an expiratory act while the glottis and the pharyngeal end of the esophagus are closed; or it would seem that the cardia is sometimes opened by some of the air being carried by esophageal peristalsis into the stomach, furnishing an opportunity for gas subjected to a higher pressure to escape. These are only the occasions of gastric belching, the expulsive force being furnished by the contractions (or elasticity if the stomach is distended) of the stomach or by intra-abdominal pressure. The peculiarity of eructatio nervosa is that these aids to belching are in whole or in part repeatedly and paroxysmally employed to relieve a distressing gastric sensation which may or may not be actually associated with an excess of gas in the stomach, and which may be relieved by the escape of gas or may persist even after the stomach

empties itself or has been emptied by a tube. Such is the double nature of the affection.

Etiology.—The affection is most common in introspective, nervous, and impressionable people. It develops in the same soil as do hysteria and neurasthenia, and hysterical and neurasthenic forms might justly be described, the attacks sometimes degenerating into feeble efforts to excite sympathy or to attract attention. Shock, anger, misfortune, great sorrows, and depressing emotions are given by patients as the occasions or causes of it. Masturbation, excessive venery, and other abuses are sometimes associated with it; indeed, many of the individuals are habit-forming neurotics or neuropaths. It is somewhat more frequent in women than in men.

The affection is sometimes associated with other diseases of the stomach, particularly, in our experience, with the displacements of the organ. In one case the attacks seemed to be associated with and dependent upon the accumulations of gas in the dilated splenic flexure of the colon. It is sometimes accompanied by gastric and intestinal "peristaltic unrest." It would seem that, apart from the displacements of the stomach, its association with other diseases of the stomach is accidental.

Clinical Description.—The affection often develops and terminates suddenly and without evident cause. The appetite, digestion, and nutrition may all be normal, but it is quite common for these patients to be emaciated and asthenic, and to complain of fullness and constriction of the stomach after meals.

The occurrence of the paroxysm has no constant relation with the taking of food or with its quantity or quality; but anger, fear, intense emotions of any kind, pressure on hysterical zones, may induce the attacks, and the peculiar causation defines a cerebrogastric group of cases. In other instances the paroxysms seem to be the quiet and natural effort to empty a displaced and flatulent stomach, or one whose pylorus is spasmodically or organically obstructed. The belching, in keeping with the genesis of the attacks, may or may not give relief.

The paroxysms recur periodically, sometimes after regular intervals, but more commonly suddenly and unexpectedly. They may continue during meals, begin after meals, or only when the stomach is in functional repose; but, as a rule, they cease at night, and never continue during sleep. An attack may begin at night. The eructated gases are chiefly those found in expired and swallowed air.

The paroxysms vary in intensity and duration. There may be a number of noisy eructations separated by a few moments of quietude; or the paroxysms may last several minutes, with ten to twenty eructations or efforts each minute; or the attacks may be severe, lasting, with short remissions, or intermissions, for several hours, or, rarely, one or two days, or even months.

The paroxysms are noisy, embarrassing, often banishing the person from society and interfering with work, uncontrollable by the will, sometimes distressing and accompanied by excitement and by anxiety, and are exceptionally followed by depression. The mild attacks have none of these serious features, and a few free eructations give complete relief.

Diagnosis.—The periodical, paroxysmal attacks, when clearly described by the patient or once observed by the physician, are characteristic. The patient is also most commonly a neurotic or a neuropath.

The complete absence of signs of organic disease of the stomach is of very great negative value. The peculiar gastric sensation, with the induced efforts to relieve it, when dissociated with other functional trouble of the stomach, is characteristic; but the discovery of abnormal functional and bacteriological signs does not necessarily exclude eructatio nervosa.

It is useless to analyze the belched gas, as this will always contain the constituents of expired air. Easier and more practical is the search for bacteriological signs—organic acids, excessive or peculiar germ growth, and gas formation in the fermentation tubes. In simple and symptomatic belching the eructated gas may be swallowed air or gas formed by fermentation or by chemical decomposition. The absence of bacteriological signs and of gastric flatulency during the paroxysm reveals the nervous nature of the trouble; but their presence does not exclude it.

Treatment.—The treatment is chiefly constitutional and is directed toward building up the nervous system. Change of scene, rest, hydrotherapy, electricity, and strong moral control and suggestion are valuable remedies. The individual himself, as well as any associated or causative disease, should be appropriately treated.

Few drugs are of value. In the non-neurasthenic cases, the bromids sometimes do good. Arsenic is seldom beneficial. Opium, belladonna, and similar drugs have proved useless in our hands.

The intragastric spray is the most valuable single remedy.

Warm water alone may be used, or warm water followed immediately after its removal by cold water, or a solution of nitrate of silver (five grs. to the pint of distilled water). The prolonged paroxysms may be cut short by 20 grs. of chloral hydrate per rectum.

VI. HABITUAL REGURGITATION.

During the course of normal digestion some of the contents of the stomach may be regurgitated into the throat or mouth. Food is then commonly brought up, in company with swallowed air or gas, and relieves a sensation of fullness in the stomach; but the regurgitated matter may also be entirely fluid. This form of regurgitation is often voluntary, or it may be a mere accident or episode of normal digestion. Pathologically, regurgitation may be a symptom of a disease of the stomach, particularly when such disease is accompanied by stagnation or retention; but regurgitation exists also as an idiopathic dynamic affection of the stomach, as a distinct morbid entity.

Clinical Description.—Habitual regurgitation is easy, involuntary, effortless, without nausea or increased salivary secretion, not preceded by conscious contraction of the stomach, and always occurs during the normal period of gastric digestion. The matter regurgitated is never solid, but always fluid or liquid, and of such a composition and taste as would be expected of the contents of the normal stomach at the moment when it occurs. The liquid rises into the throat or mouth, and not simply into the esophagus, and is either expectorated or is swallowed again through natural feelings of delicacy. It is never remasticated with enjoyment and again swallowed, as in rumination.

The regurgitations recur quite regularly after each meal, are several times repeated, and are noteworthy in quantity. Rarely, the quantity of food lost by spitting it out is so great as to produce inanition and to confine the weak patient in bed. This severe form is likely to be confounded with habitual vomiting.

The regurgitation may often be suppressed by a strong effort of the will or by swallowing at the moment when it begins. Occurring, when not resisted, without discomfort, when voluntarily prevented it is usually accompanied by a sensation of fullness or distention in the lower part of the esophagus.

Diagnosis.—Regurgitation will not be confounded with habitual vomiting when the distinctive characters already given are noticed; but it may be mistaken for esophageal regurgitation, particularly in esophageal pocketing and the stagnation or retention of esophageal stenosis. The matter regurgitated from the esophagus has never entered the stomach, and consequently contains neither free nor organically combined HCl, nor gastric ferments, nor products of gastric digestion. The esophageal regurgitated matter often contains pus, and consists largely of mucus, sometimes foul or fermenting; the gastric contents, having markedly contrasting and characteristic properties, can be obtained through a tube introduced into the stomach. The differentiation is easy after suspicion is once aroused and the proper explorations are made.

Treatment.—The treatment of habitual regurgitation is both general and local. Any associated trouble should receive attention, and an attempt should be encouraged to break the habit by force of will and by its voluntary suppression. The general health should be improved, and the nervous system particularly should be strengthened by hygienic and physical remedies. The bromids may prove to be of some value. Strychnin is more trustworthy. Intragastric faradization is beneficial, or cervicogastric galvanization may be employed, particularly when the regurgitation is due to insufficiency of the cardia. More important is the regulation of the diet, selecting those articles of food which leave the stomach earliest and excite its functions least. All the food should be most minutely divided. Constipation, rapid eating, and fatigue increase the trouble.

VII. RUMINATION, OR MERYCISM.

Rumination is a motor disorder of the stomach characterized by the easy, quiet, effortless, sometimes voluntary, sometimes involuntary, regurgitation into the mouth of food which is (according to its taste and to the mental peculiarities of the patient) at times spit out, and at times reswallowed after a second mastication, which, instead of exciting disgust, is performed with pleasure. Pyrosis, regurgitation, and rumination differ more in degree than in nature—out of the voluntary act the habitual and involuntary may develop. Remastication is a distinctive and essential characteristic of rumination, but it is not present at all times in a particular case. In

merycism man really "chews the cud," as do the ruminants, but only a part of the food regurgitated may be remasticated and swallowed, the remainder being spit out; or remastication may be only occasionally performed. It is the second mastication of the food which distinguishes rumination from regurgitation, and without its detection the existence of the affection can not be established. Habitual regurgitation may be just as obstinate and persistent.

Pathogenesis.—Various explanations of the nature of rumination have been given. It has been supposed to be due to paresis of the cardia; but the deglutition sounds are normal, the gas and air do not escape when the stomach is inflated, and it is hardly probable that the cardia is paretic while the rest of the stomach is normally or excessively active, or that it can be made paretic by irritation. The regurgitation has been explained as being produced by irritation of the vagus (periphery or center) and by the active opening of the cardia through Openchowski's dilator fibers of the cardia; or by reflex relaxation of the cardia; or it has been supposed to be produced in the same manner as voluntary belching. It would seem at times that the regurgitation is an uncontrollable habit, which was voluntary in its beginning.

This motor dynamic affection of the stomach occurs in all sorts and conditions of men. It is very frequent among idiots and the insane. Indeed, the remastication without shame and with even positive enjoyment presupposes a certain degree of mental weakness. The ancients, not without some show of shrewdness, supposed that a remasticating man had in some way become possessed of the nature and instincts of the cud-chewing animals. Rapid eating, habitual regurgitation, and imitation are occasions of the ruminating habit. The motor function of the stomach becomes subservient to a perverted head. It often stands in close relation with the mental affection, developing and declining with it and disappearing during lucid intervals. Merycism is more frequent in men than in women, and may or may not be associated with other diseases of the stomach.

Clinical Description.—The regurgitation is confined to the period of gastric digestion. During the period of repose it is rare that the gases and the small quantity of secretions in the stomach are voluntarily or involuntarily brought up into the mouth. Water or coffee taken into an empty stomach is not regurgitated, but wine, beer, and other alcoholic drinks are frequently brought up and reswallowed. This rule is, however, not without exceptions. The regurgitation

is often selective, the unmasticated solids being usually brought up—making plausible the opinion of one of our patients who spoke of rumination as a "beautiful provision of nature for the protection of the digestive organs against rapid eating." The regurgitation occurs without noise, effort, or discomfort, and the remastication continues as long as the contents of the stomach have a pleasant taste.

The procedure usually begins immediately after the meal, and is continued as long as any pleasure or supposed advantage is derived from it. The regurgitated food is either spit out or reswallowed as soon as it is perceived to be sour or unpalatable. Consequently, remastication is most frequent during the first half hour of digestion, but may continue longer or begin later, particularly where secretion is inactive and the food eaten is such as remains long in the stomach.

The body is usually well nourished, but emaciation occurs, and may become extreme where much of the regurgitated food is not reswallowed. The state of nutrition is largely dependent on the functional integrity of the intestines, the meats and albuminous foods being often imperfectly digested by the stomach in rumination. Salivary digestion, on the other hand, is unusually active.

The duration of the affection is indefinite, sometimes beginning suddenly and unexpectedly ceasing, sometimes intermittent, but more frequently obstinate and persistent from early youth to old age.

Rumination may occur independently or may be associated with other diseases of the stomach. It usually ceases when a painful affection or a severe disease of the stomach develops, and the gastric symptoms which follow its disappearance are due, not to the suppression of the rumination, but to the disease of the stomach which caused its cessation. In one of our cases it ceased with the sudden development of pyloric incontinence following a heavy financial loss. Hydrochloric acidity may be normal, increased, or diminished, and delayed hyperchlorhydria is frequent; the evacuation of the stomach may be normal, delayed, or too rapid. There appears to be no fixed relation between rumination and the states of secretion and of the motor function. The stomach may be normal in size, enlarged, or very small. In a case of the authors' the stomach was very small, holding about twelve ounces when full, indistensible, the greatest transverse diameter being about 2½ inches. The mucous membrane was atrophied. The patient had ruminated persistently for fifty years. In still another case the rumination began farther

back than the memory of the aged patient could go, and the rumination suddenly ceased two years before the death of the patient, which was due to a disseminated hard cancer that converted the pylorus into an incontinent ring or open canal.

Rumination has one pathognomonic sign—the remastication of the regurgitated food. It is always digestive, which is not true of esophageal regurgitation. In habitual gastric regurgitation and in vomiting the food is never remasticated. The beastly enjoyment of cud-chewing is definitive, and without this sign habitual regurgitation and rumination can not be differentiated. In merycism the regurgitated food, on account of its unpleasant taste, may be spit out or reswallowed.

Treatment.—The treatment of rumination is sometimes successful. The patient should be persuaded, if possible, of the disgusting unnaturalness of the habit, and the will should be engaged in an effort to suppress the regurgitation or to reswallow immediately the regurgitated food without a second mastication. Thorough mastication of the food and the selection of a diet that rapidly leaves the stomach are prophylactic measures. The administration of hydrochloric acid, or, in suitable cases, of alkalies, may be beneficial. Strychnin and quinin destroy the insane delight of remastication, and may be used if they will induce the patient to reswallow the regurgitated bitter food. Electricity and strychnin may be employed in the treatment of the secondary paresis of the cardia and the secondary dilatation of the lower part of the esophagus which sometimes exist.

VIII. NERVOUS VOMITING.

Every act of vomiting is nervous, but the incrimination of the nervous system would be manifestly unjust when it is responding to excitation in a normal manner. Vomiting as a dynamic affection of the stomach is not only produced by the nervous system, but is also the expression of a particular state of the nerve-centers that govern the movements of the stomach. From the influences of these centers on the vomiting center located in the medulla result the co-ordinated contractions of the voluntary muscles concerned in the production of the overt act.

The vomiting center is supposed to be represented by a distinct nucleus of one of the roots of the vagus, and is in close eccentric relation with the centers that transmit motor

impulses to the muscles of the thorax, abdomen, diaphragm, larynx, pharynx, esophagus, and stomach. Through this center the complex act of vomiting is excited and performed. The contracted abdominal muscles and diaphragm compress the stomach. The stomach, particularly the pyloric part, contracts, antiperistalsis begins, the pylorus closes, the cardia opens, the esophagus is shortened by contraction of its longitudinal fibers, the glottis is closed, the pressure is removed from the esophagus by expansion of the thorax, the soft palate shuts off the rhinopharynx, and the contents of the stomach are forced in successive streams through the mouth. All these parts are not essential to vomiting. The stomach may be passive, as in gastroplegia. The pylorus may be open, as in pyloric incontinence. Vomiting may be effortless, and may occur at the end of expiration and before either the glottis or the rhinopharynx is closed. It may occur in spite of strong voluntary effort to suppress it. But, however performed, the act is excited and controlled by the center of vomiting in the medulla.

Very complex indeed are the relations of this center to the various parts of the organism, and correspondingly numerous are the causes of its activity. It may be directly irritated by disease of the medulla, or indirectly by disease of associated cerebrospinal centers, by an abnormal blood or blood-supply, and by impressions reflected from various organs. All these forms of vomiting are symptomatic, and must be excluded before the diagnosis of nervous vomiting can be made.

1. SYMPTOMATIC VOMITING.

Central Vomiting.—The morbid causes of central vomiting may be located in the brain, medulla, or cord and in their membranous or bony coverings. It is a common symptom of meningitis, embolism, thrombosis, apoplexy, abscess, traumatism of the brain, hydrocephalus, and cerebral tumors. The last is one of the common causes of cerebral vomiting. Irritative lesions of the medulla and exophthalmic goiter, multiple sclerosis, compression myelitis, and particularly tabes dorsalis, may also produce it. In all cases of recurring or persistent vomiting without apparent cause the signs of organic disease of the central nervous system should be diligently sought, and an ophthalmoscopic examination should be made.

Hematogenous Vomiting.—The activity of the vomiting center may be excited by a deficiency of blood or by toxemia.

Anemia, fainting, and prostration are sometimes accompanied by vomiting. Tobacco, opium, chloroform, ether, and other narcotics exert a toxic action on the medulla, as do also forms of uremia, of sepsis, and of intestinal auto-intoxication. The sudden entrance of bacterial poisons into the system may also excite vomiting, and in this respect the poisons of cholera, of malaria, and of scarlet fever seem to be particularly active. Vomiting should not be called nervous until these causes have been excluded.

Reflex Vomiting.—Reflex vomiting is the most common form and is produced by a very large number of diseases of the various organs. It is a symptom of disease of the labyrinth and, rarely, of the middle ear. Nasal and pharyngeal tumors may cause it. It is quite common in whooping-cough and in laryngeal tuberculosis. Disease of the lungs, and, more rarely, of the pleura, may cause it; it is sometimes very obstinate in pneumonia, and it is almost a clinical axiom that one who coughs after eating and vomits after the coughing is a consumptive; but the most frequent sources of reflex vomiting are the abdominal organs. The diseases of the stomach need only be mentioned. Cholelithiasis is often an obscure cause. Nephritis, pyelitis, calculus, and displacement of the kidney should not be forgotten. Pregnancy and diseases of the uterus and its appendages are frequent causes of reflex vomiting in women. The diseases of the intestines probably produce vomiting even more frequently than those of the stomach itself. Among these may be mentioned enteritis, colitis, appendicitis, ulcer, obstruction, constipation, neoplasms, and intestinal worms. Peritonitis is still another abdominal cause of reflex vomiting.

Before the diagnosis of purely nervous vomiting is made, the diseases of which vomiting is a symptom should be excluded as far as possible by a thorough and complete examination. And if such disease be discovered, the diagnosis is not sure, for the question whether vomiting is an associated dynamic affection or a symptom of an associated disease of the stomach remains to be answered.

2. NERVOUS VOMITING.

Nervous vomiting as a dynamic affection occurs in three distinct clinical forms—hysterical, psychic, and periodical.

Hysterical vomiting is rarely encountered except in the female sex, and is very variable in its clinical characters. Occasionally, the nervous vomiting is the only manifestation

of the hysteria, or it may follow or alternate with other symptoms of this protean affection. Its nature is often revealed by the conduct of the patient (posing, longing for sympathy or for attention, perfect freedom from anxiety), by the irregularity of its occurrence, and by its peculiarities in regard to the quality and quantity of the food. It is always digestive and the vomit is always alimentary. It comes and goes intermittently without evident cause. Only solids, or only liquids, or only a particular food may be vomited. There is no other discoverable disease. The evacuation of the stomach is ordinarily incomplete, and there may be but slight loss of weight and strength.

A more common form of hysterical vomiting is associated with inactivity of all the vegetative functions. Less oxygen is consumed, less carbonic acid exhaled, and less heat is formed than in health. The skin is dry. The quantity of urea and of all the other excrementitious constituents of the urine eliminated during the twenty-four hours is notably diminished. There may be almost complete anuria. The nervous symptoms of uremia are absent, and there are no signs of disease of the kidneys and no obstruction of the ureters. The body, in spite of the vomiting and of the apparently serious insufficiency, remains well nourished, on account of the inhibition of nutritive exchange. There may be urea or ammonia in the vomit, the gastric intolerance may be complete, but the system shows no other signs of self-poisoning. The trouble may last for days, and may disappear as suddenly and unexpectedly as it began, or may be followed by other hysterical manifestations.

Psychic vomiting is the most common of the three clinical forms of the dynamic affection, and is either mild or severe. It is usually the result of emotive shock or of mental overwork—intense fright, anxiety, sudden misfortune. It is most frequent among pale and overworked school-children, but may be encountered in all walks of life and in all occupations. It sometimes affects men suddenly thrust into responsible positions.

In the mild form more or less of the food eaten is periodically vomited, or the incomplete vomiting may occur after each meal. The loss of food is not sufficient notably to impair nutrition. Long or short in duration, it usually ends suddenly after the cause has been removed.

The severe form is also characterized by alimentary vomiting, copious but often capricious. Inanition is the result of the inability to retain sufficient food, and, exceptionally, the

disease may be fatal. But an intercurrent disease usually cuts the thin-spun thread before death from starvation occurs. The uncontrollable vomiting and the resulting inanition are the only symptoms, the disease getting its name from its mode of origin.

Periodical vomiting is a rare but a severe form of the dynamic affection. It is characterized by periodical attacks of complete gastric intolerance, which are separated by intervals of perfect health. The intervals in the same case are always of nearly the same length, during which time no disease of the nervous system nor of the stomach nor of any other organ can be recognized. The attacks represent periodical breaks in the course of good health, and begin and end suddenly and in an apparently causeless manner. The duration of the interval is from several days to several months, and that of the attack from several hours to several days, but both the interval and the attack have a definite and constant duration in each particular case.

The disease begins with vomiting, frequently in the morning before breakfast, and without warning, or preceded by a slight headache and gastric discomfort, and sometimes nausea. The vomiting occurs repeatedly, both spontaneously and after the ingestion of food, drinks, or drugs. The intolerance of the stomach is complete, the vomit in the beginning consisting of the contents of the stomach, and thereafter of the secretions of the stomach and of the duodenum and its accessory glands,—mucus, bile, pancreatic and gastric juice,—mixed with saliva and whatever has been introduced into the stomach. There may be no pain except that due to retching, but severe cramps sometimes occur, along with muscular pains in the lower extremities. The patient soon becomes anxious and prostrated. The abdomen is depressed, but the abdominal muscles are often soft. Beneath lie the contractured stomach and intestines, alike intolerant of distention or of interference. Constipation is obstinate, and an enema is badly taken, and the little water introduced is quickly expelled. The end of the attack is as sudden and causeless as its beginning. There is no constant nor characteristic disorder of gastric secretion.

The periodicity is not a pathognomonic sign, as vomiting often occurs periodically as a symptom. Periodical symptomatic vomiting may be the first and only sign of locomotor ataxia, or the expression of any of the forms of central, of hematogenous, and of reflex vomiting. The periodicity, the character and evolution of the attack, the absence of any

disease that could cause the vomiting, the intervals of perfect health, are the distinctive features. Emaciation is the result of frequent and severe attacks, and the disease may be fatal.

The diagnosis of this rare form of nervous vomiting should be made only after the most exhaustive and repeated examinations. Time only can exclude incipient tabes. The vomiting of migraine marks the end of the attack, whereas periodical vomiting begins without warning.

Diagnosis.—The diagnosis of the cause and of the particular form of vomiting may be easy or very difficult. It is best always to proceed with the investigation in a methodical manner. Is the vomiting due to a disease of the stomach? If not, is the vomiting symptomatic or nervous? And if nervous, what is the particular form?

If the vomiting is due to a disease of the stomach it will be accompanied by the usual symptoms and signs of that particular disease; consequently, the clinical history and the physical, functional, bacteriological, and anatomical signs obtained by the thorough examination of the stomach may at once reveal the particular disease. Or, in case the result of the examination is negative, it remains to be determined whether the vomiting is symptomatic or nervous. But the decision is not to be based on the result of the examination of the stomach only, for there are other symptoms and signs which positively suggest or reveal the symptomatic or nervous character of the vomiting. There may be subjective and objective evidences of disease of another organ clinically known to cause vomiting, having the same distinctive features as the case under investigation. The vomiting may occur easily, painlessly, and without discoverable gastric cause, when the stomach is empty; or may be dependent on the state of the feelings or of the mind—in both of these conditions the vomiting is not likely to be due to disease of the stomach. Food may be tolerated that is known to be borne with difficulty, or may be vomited when it would be rationally expected to agree better than food that is retained. Diet alone, in symptomatic and nervous vomiting, is likely to be of little benefit, but a correct diet often arrests at once the vomiting due to a disease of the stomach, and in both symptomatic and nervous vomiting the exclusively gastric medication is of no value. The indifference of the patient and the maintenance of good nutrition, in spite of the absorption of but little nutriment, would reveal its nervous character.

After excluding a disease of the stomach, it may be difficult or impossible to decide whether the vomiting is sympto-

matic or nervous. Symptomatic vomiting is caused by disease, and on careful examination this disease can, as a rule, be detected. If the disease is such as usually excites vomiting, if the gastric disturbance is greatest during the period of functional activity of the diseased organ, and if the vomiting is benefited by the proper treatment of this disease, it is fair to conclude that the vomiting is symptomatic. Nervous vomiting is always afebrile. Hysterical, psychic, and periodical vomiting possess certain clear-cut features that often suggest at once the correct diagnosis. There is no doubt that nervous vomiting, in the proper and limited meaning of the word, is much rarer than is commonly supposed, and becomes less and less frequent as our methods of investigation gain in completeness and precision.

Treatment.—The treatment of all forms of vomiting is dominated by three principles—the control or removal of the source of excessive irritation ; the prevention of the action of the cause on the medullary center of vomiting or the arrest of impressions sent out from it; and the maintenance of the repose of the stomach. Or, more briefly stated, the three objects of medication are to control or remove the cause, the transmission, and the expression. The treatment of nervous vomiting is based on the same principles.

The cause of nervous vomiting is not always palpable, but the influence of the mind and of the moral environment is often very evident. The physician can do a great deal, by gentle authority and by assuring suggestions, to relieve the general demoralization. The hysterical should be controlled by a firm and skilfully directed hand, and moral and mental repose and balance should be restored by whatever means a knowledge of human nature will suggest as most applicable to the individual case. Isolation, change of scene, and cessation of study are often necessary. Absolute and continuous rest in bed may be demanded for the restoration of nervous strength and the equalization of the circulation, and the patient should be kept in bed at least during the attack. Massage, electricity, and hydrotherapy, in forms suitable for the particular case, may often be employed with benefit. By these means and by attention to all the vegetative functions, the nervous system may be given new vigor and tone and the transmission of the cerebral impulses may be prevented.

Something may also be accomplished by gastric medication. Electricity, hydrotherapy, and a proper diet are our best remedies. During the intervals or during the best moments a simple, nutritious, and mixed diet should be

ordered, and in suitable cases gavage (overfeeding by means of the stomach-tube) may be employed.

The foregoing medication is the basis of the curative treatment. The vomiting itself, however, often requires palliative remedies.

Menthol, chloroform, iodin, and the bromids are rarely of much value. Oxalate of cerium, in five-grain doses dry on the tongue, is trustworthy and sometimes efficient. The dried alcoholic extracts of coca (three grs.), of kola (five grs.), and of belladonna ($\frac{1}{10}$ of a gr.) sometimes act efficiently when in combination. A tablet may be allowed to melt on the back part of the tongue or the mixed powder may be placed there. Neither these drugs nor the oxalate of cerium act so efficiently when swallowed with water. Suppositories of asafetida (five grs.) and powdered extract of valerian (three grs.), repeated every two hours, exert a very soothing and quieting influence. If there be much prostration, strychnin hypodermically is the best palliative; or, if there be much excitement, the phosphate of codein may be given hypodermically, or chloral hydrate by rectum, or morphin may be at once used, without trial of less trustworthy remedies. Nitroglycerin may be useful to equalize the circulation.

Sedative galvanization is a remedy well worthy of trial in the obstinate cases. Large electrodes should be placed over the stomach (anode) and over the cervical spine, and a current of low density should be slowly turned on and slowly cut off after being allowed to flow uninterruptedly for from ten to twenty minutes. Or sedative polar cervicogastric galvanization may be used, or even intragastric faradization.

An excellent remedy is cold or heat applied to the spine and to the epigastrium. The rubber coils are most convenient, and through them either ice-cold or hot water may be allowed to flow. Heat is best in prostration and cold in excitement; but the idiosyncrasy of the patient should be considered. The ether spray may be used along the spine and over the epigastrium when a quick effect is desired. A blister (canthos plaster) over the back of the neck and over the epigastrium is sometimes accompanied by the cessation of the vomiting.

If the stomach is intolerant, the patient should be kept perfectly quiet, in the recumbent position, and neither food nor medicines should be given by mouth; rectal feeding is then our best resource. Hysterical intolerance is often relieved by the introduction of the food into the stomach

through the tube, the food thus introduced being generally retained. In suitable cases overfeeding with the tube (gavage) may be tried.

In the milder cases the diet must be selected by clinical experimentation, there being in the absence of digestive disease no contraindication to any food, and the foods difficult of digestion are sometimes best borne. Often in severe attacks small quantities of dry, solid food can be retained when liquids are at once rejected. If there be much prostration the patient should be kept strictly and persistently in the recumbent position. The treatment, as it thus appears, varies with each individual case, and many of the foregoing remedies are generally applicable.

IX. INCONTINENCE OF THE PYLORUS.

Contrary to the common opinion, the pylorus is, in all probability, lightly closed during the period of repose of the normal stomach; for gas remains in the viscus in spite of its retraction, and reflux of bile and duodenal secretion does not occur. The passage is more easily forced by gentle pressure from the duodenal than from the gastric side (Oser). Stimulation of the sympathetic relaxes, but that of the vagus closes it. But it is more likely that normal contraction and relaxation of the pylorus during the period of gastric digestion are automatically regulated through the ganglia in its walls.

The pylorus is incontinent when it is insufficient during digestion, neither controlling nor preventing the too rapid evacuation of the stomach. The trouble may be organic or functional.

Pathogenesis.—The organic form occurs in cancer, ulcer, and duodenal obstruction. Carcinoma, particularly scirrhus, may convert the pylorus into a rigid tube with an unobstructed lumen, or the canal may be made by ulceration of the neoplasm, or the ring muscle may be rendered stiff and functionless by cancerous infiltration. Ulcer, when either lateral or circular, may also destroy the contractility of the muscle, or a cicatrix or a cicatricial band or adhesion (perigastritis) may hold the canal open. In duodenal stenosis, or even in stenosis of the jejunum, the pylorus may be mechanically dilated by the material that accumulates above the obstruction. Gastroptosis with angular constriction of the duodenum may be associated with pyloric dilatation. But either the history or signs of ulcer or of cancer or of duo-

denal obstruction will reveal the organic nature of the pyloric incontinence.

Very rarely incontinence of the pylorus is a dynamic affection. Quite frequently the stomach empties itself with abnormal rapidity, but most commonly on account of excessive peristalsis. When the rapid evacuation is due to the insufficiency of the pylorus, it is not prevented by drugs that control peristalsis.

Clinical Description.—Pyloric incontinence, except when due to duodenal obstruction, is manifested by a few symptoms that may be somewhat characteristic. It is sometimes noticed that the abdomen, previously flat, suddenly becomes tympanitic after eating; but this symptom occurs in hysteria, and may be explained in other ways. On artificial inflation the stomach does not fill and distend and outline itself on the abdominal wall, but with each pressure of the bulb, or contemporaneously with the generation of carbon dioxid, pyloric bubbling can be palpated and heard, and the limitation of the stomach by percussion becomes impossible. Eventually the stomach may be distended with the air, but its limits can not be determined by percussion. The inflation test is of more value when it is made during digestion, when the stomach is easily inflated if the pylorus is not insufficient; if gas bubbles through the pylorus, the bubbling occurs in relation with the compressions of the inflating rubber bulb, the latter sign, under the circumstances, being characteristic of pyloric incontinence. In hypermotility inflation is easy and the evacuation of the stomach is peristaltic. These positive or negalive peculiarities of inflation are distinctive when they occur with regularity and in association with the following signs.

The stomach persistently empties itself too rapidly after the test-breakfast, after the test-dinner, after a glass of milk, and after two glasses of water have been taken. Contrary to the distinctions that exist normally, one food leaves the stomach about as rapidly as another. As a consequence of this phenomenon, lienteric diarrhea is common when the diet is exciting and unsuited to the intestines. A bland food like milk rapidly cures this particular form of diarrhea. Intubation of the pylorus is less difficult than when the pylorus normally contracts.

The reflux of bile and pancreatic juice, as would naturally be expected, is easy, but it does not occur constantly. Previous eructation, regurgitation, rumination, and vomiting usually cease when the pylorus becomes incontinent; but this is not always so.

Diagnosis.—The diagnosis is made by the foregoing group of signs and symptoms in the absence of all evidence of ulcer, cancer, or hypermotility. The trouble is not dangerous, as is conclusively proven by the results of pyloroplasty and pylorectomy.

Treatment.—The treatment is chiefly dietetic, such food being prepared and selected as is readily digested by the intestines. Diarrhea and intestinal colic may necessitate frequent small meals. Strychnin and hydrastinin may be of some use, but intragastric faradization is more rational. In cancer and ulcer the incontinence of the pylorus is an advantage.

X. GASTROPLEGIA.

Paralysis of the stomach is rare and has usually been confounded with "dilatation." It is a distinct affection, characacterized by the sudden loss of the contractile power of the muscular layer. In myasthenia the muscle is weak and has lost more or less of its normal elasticity and tone. In gastroplegia the muscle is paralyzed and the power to contract is lost. The stomach is a motionless sac that passively yields to mechanical stretching.

The chief cause of gastroplegia is traumatic shock. After an accident, or particularly after a laparotomy, the stomach is found to be paralyzed. The same condition may occur in hysteria and as a result of moral shock during the digestion of a meal. Still another cause is acute gastritis, and it may occur in multiple neuritis.

The beginning is sudden and the duration variable. After a few days the muscle may gradually regain its power, or the loss of contractility may be permanent. After laparotomy and traumatism it may be accompanied by collapse.

The chief sign is absolute gastric retention. Food and secretions and gases accumulate in the stomach and render the epigastrium prominent. The distention of the stomach is painful, and the contents are often partly removed by expression and by overflow through the pylorus.

The stomach should be kept clean and empty by lavage and the body should be nourished by rectum. A part of the treatment is that of shock and of the causative disease—hysteria, peritonitis, neuritis, etc. The ordinary means should be employed to restore the paralyzed muscle or prevent its atrophy. The sovereign remedy of traumatic gastroplegia is strychnin, administered hypodermically in large doses.

CHAPTER IV.

NEURASTHENIA GASTRICA.

NEURASTHENIA (*νεῦρον*, nerve, and *ἀσθενέια*, weakness) gastrica is a dynamic affection characterized by excessive irritability and marked weakness of the nerves which supply the stomach, branches of the pneumogastric and of the solar plexus. It is a particular kind of morbid sensibility of the stomach, developing in a fit constitution after the excessive expenditure of nerve force. It is digestive discomfort without noteworthy modification of the process of digestion and without an anatomical lesion of the mucous membrane.

Gastric neurasthenia is not limited to the cases where the uncomfortable sensations and the other symptoms of which the patient complains are referred to the stomach. The manifestations of the irritable weakness of the nerves supplying the stomach may occur in distant parts of the body. It is not the localization of the expression, but that of the weak and genetic point which defines the affection. It is essentially a dynamic sensory affection, and, more definitely, an affection chiefly of the abdominal sympathetic.

The solar plexus, receiving all the impressions from the abdominal and thoracic organs, is very intimately associated with the cerebrum. Through it sensation, thought, and emotion influence digestion. Through it and the pneumogastric nerves digestion affects the activity of the brain. Through it most of the distant nervous symptoms of the diseases of the stomach are transmitted. It is the connecting link between the moral, the intellectual, and the vegetative life. And this is best seen not in health, when its working is silent and invisible, not in experimentation, when its action is imperceptible or unphysiological, but in disease, when its morbid action rises into consciousness and is expressed by certain symptoms and signs. It is this highest and greatest assemblage of sympathetic centers which unites the nervous symptoms of neurasthenia gastrica.

Strictly localized in the beginning, the irritable weakness extends to the connected cerebral and spinal centers, or, originating in them, secondarily affects the solar plexus. The vicious circle thus becomes established. The clinical picture

varies accordingly as the cerebrospinal or abdominal centers control the generation of the manifestations; but even when the gastric symptoms predominate, their variation, with the mental and moral changes, may be easily observed.

The peculiar discomfort of neurasthenia gastrica is not due to any anatomical lesion of the mucosa, and is out of all proportion to the disturbance of the digestive functions. This is one of its cardinal and distinctive characteristics. Not all gastric symptoms are neurasthenic—not even when they are of the same character and grouping as in this affection. Digestion often becomes uncomfortably perceptible in other diseases, but the effect stands in a natural proportion to its cause. When the irritation is abnormal a corresponding effect should naturally follow. The harmony is preserved between the activity of the external cause and the intensity of perception. In neurasthenia, on the other hand, this harmony is lost, and the effect is many times multiplied. The nerve-centers are feverishly active. The perceived and actual effect is excessive—too intense and too prolonged. While the action is excessive, the total energy is less; but the excessive action is not sufficient to produce gastric spasm, nor does the weakness amount to paresis. Secretion may remain normal, the mechanical work may be efficient, or both may be excessively active—but digestion rises painfully into consciousness. Secretion may diminish slightly, the motor function become insufficient, or both may continue needlessly long—still digestion is distressing. The patients suffer, be the evolution of digestion normal, hastened, or delayed. The want of harmony between cause and effect exists; the impressions, normally unfelt, are multiplied and built up into conscious sensations on account of the irritable weakness of the nerve-supply of the stomach.

Etiology.—Neurasthenia gastrica is common in both sexes during adult and middle life. A large number of the cases occur among students, musicians, and teachers, and among those on whom the cares of life and the reverses of fortune have fallen heavily. Worry and overwork lay the foundation for a majority of the cases. Prolonged and excessive expenditure of nerve force, particularly by sexual excesses and abuses, and by unsatisfied sexual excitement, exhausts and overexcites in a marked manner the abdominal sympathetic.

The irritable weakness of the nerve-supply of the stomach may be developed in a number of ways. A nervous temperament and constitution, inherited or acquired, predispose

to it. Neurasthenia gastrica is often associated as cause and effect with general neurasthenia. The exhausting and irritating impressions may come from the brain or may be reflected from other organs. The neurasthenia may be partly caused by a blood state, as uricemia, uremia, diabetes, auto-intoxication, or by a blood disease. The anatomical diseases of the stomach often produce it, but it is then only secondary and symptomatic, and not a distinct affection. The excitations which occasion the outbreak when the nervous system is already weak may originate in the mucous membrane of the stomach, from irritant foods, drinks, and drugs; but the most important factors in the genesis of neurasthenia gastrica, in our experience, are disease of the intestines, sexual excesses, self-abuse, worry, and overstudy.

Clinical Description.—All the symptoms of neurasthenia gastrica are referable to the irritable weakness of the nerve-supply of the stomach. These nerves and their centers may be alone involved, and may produce the whole clinical picture, or they may be affected in common with the general nervous system, and may directly cause only a part of the manifestations. In four-fifths of the cases of all forms of neurasthenia the gastric symptoms become prominent at some moment in its evolution. So frequently do the symptoms of neurasthenia, be the affection primary or secondary, cluster about the solar plexus.

The affection is variable in its severity and in its evolution. The course may be intermittent, remittent, or stationary; or it may rapidly progress from a very mild beginning to a mental, moral, physical, and nutritive state sufficiently severe to excite alarm. In the mild cases the patients are only diseased because they complain, there being no objective signs of trouble. The nutrition, strength, and appearance of health are preserved, the digestion and utilization of food are normal, all the organs, including the stomach, perform their functions physiologically. The affection may be purely subjective, the patients searching in vain for words strong enough to describe their sensations and to persuade others of the reality of their sufferings. In the very severe cases, however, the objective signs may alarm the physician. Emaciation and loss of strength may suggest a severe anatomical disease. The appetite may be lost, the diet restricted and insufficient, insomnia obstinate, and the secretory and motor functions of the stomach and intestines may be depressed. The subnutrition is the result not of an insufficient diet alone, but of the influence of the peculiar discomfort and of the mental, moral, and

physical state of the patient on the functions of the stomach. Between the mild and the severe cases all grades of the affection exist, some simple and others complicated.

The symptoms differ during the period of digestion and the period of gastric repose, are markedly influenced by the mental and moral state, are commonly associated primarily and eventually with some of the signs of general neurasthenia, and the suffering, shorn of all acuteness, is moderate, unnerving, and sympathetic. The characteristic symptoms are sensory, cerebral, muscular, and circulatory.

The sensory symptoms are the hyperesthesia and neuralgia and the indefinable discomfort due to the excitation and irritable weakness of the abdominal sympathetic. During digestion, and not rarely when the stomach is in repose, there is a sensation of fullness, heaviness, or weight. Whatever touches the mucous membrane—food, gas, drinks, secretions, contact of the two surfaces—may produce distress. Belching is common, and the patient often complains of a ball in the throat or gullet and of heartburn which is due to an excess of neither hydrochloric nor butyric acids. Or the patient complains chiefly of a peculiar digestive uneasiness and discomfort, which may begin as soon as food enters the stomach, or after a short period of exaltation and well-being, or which may be confined to the period of free acidity. This free acid discomfort may develop into severe pain, which is immediately relieved by bland nitrogenous food of a high acid-combining power, like milk. There is no excessive acidity and the appetite is not increased, but often diminished, thus distinguishing the symptom from adenohypersthenia gastrica and bulimia. The skin over the stomach, both before and behind, is often abnormally sensitive. The hyperesthesia is over the area supplied by the cutaneous sensory nerves connected with the irritated part of the sympathetic system. There are often epigastric, dorsal, and intercostal neuralgia and painful pressure-points. The sensitive points correspond, usually, with the large sympathetic ganglia and with the sensory branches of the spinal nerves along either side of the dorsal spine. The hyperesthesia of neurasthenia gastrica is characterized by its beginning over the stomach, where it is most constant, and extending to the sides and thorax, neck, and shoulders, and often to the head. It is often confined to the upper part of the left side of the body or head.

Normally, during the digestion of a full meal there is a desire for repose and isolation, but the depression of the cerebral functions may be prevented by stimulants like coffee,

tobacco, and entertaining companionship. Digestion physiologically enforces cerebral inactivity. But in neurasthenia gastrica the natural depression may be supplanted by a short period of well-being, or may be immediately followed by disordered cerebral activity and unhealthy sensations. The head is heavy or light, the thoughts ramble, ideas follow one another in disorder, and the consciousness of this confusion produces anxiety, gloom, and fear. The anxiety has no external cause and the fear no external object—both are centrally excited. Study and concentration of attention and control of thought are difficult, and often become impossible after a short effort. The mind is rapidly drawn aside in spite of the will. There is no repose of mind or spirit, but a constant internal unrest. The patient is made anxious and pessimistic by little accidents which would have no effect in health. The physician's assurance that there is no serious disease is as nothing when weighed against the suffering, unrest, and utter lack of energy and will-power. These cerebral symptoms are often accompanied by headache, insomnia, and vertigo, are worse during digestion, and subside slowly after digestion is finished if there be no secondary sources of irritation.

The contents of the stomach irritate the oversensitive nerves of the mucous membrane, and these impressions are transmitted to the solar plexus and the medulla. From these centers the heart's action and the vasomotor nerves often become disturbed. Tachycardia, palpitation, *bruit de galop*, arrhythmia, intermittent pulse, hot flashes, and cold hands and feet result. The involuntary muscles become weak and irritable, and the voluntary muscles may be easily exhausted.

All these symptoms vary in their intensity from day to day, and are rarely all present in the same case. Neurasthenia gastrica may be monosymptomatic in its expression—digestive discomfort, or disturbance of the action of the heart, or neuralgia, or pessimism, or headache, or other cerebral symptoms. Their intimate relation to digestion is a distinctive characteristic of these neurasthenic symptoms.

In some cases the discomfort and flatulency are excited by certain articles of food, particularly acids and sweets, in contradiction to the general rule that in neurasthenia gastrica one food is digested about as comfortably as another—no better and no worse. Guided by experience, one food is excluded after another, until some patients become meat eaters, or vegetarians, or half starve themselves.

The functions of the intestines are rarely normal in neuras-

thenia gastrica, because the whole abdominal sympathetic is often in the same state of irritable weakness as the solar plexus and the ganglia in the gastric wall. The abdomen is sometimes sunken and the intestines contracted and empty, particularly in the severe form with subnutrition. Sometimes there is nervous diarrhea, sometimes general gaseous distention of the intestines; but the most characteristic condition is localized contractions and isolated gaseous distention of individual knuckles of the intestines. The most frequent sites of this gas accumulation are in the cecum, the lower end of the ileum, and in the transverse colon near the splenic flexure. The patient complains of the gas remaining still, not easily passing either up or down. These distended knuckles are always tender, and the gas gurgles under moderate pressure and does not return to the point after the removal of the pressure. After the gas is pressed out the tenderness disappears. Massage restores, in this condition, the normal peristaltic flow of the contents more rapidly and efficiently than in any other intestinal affection, and is of great utility in relieving the radiated nervous symptoms and in establishing the diagnosis.

In the majority of the cases of neurasthenia gastrica secretion is normal, but in the remainder of the cases the acid, and rarely the ferment, secretion, may vary slightly from the normal. There is, exceptionally, mild supersecretion; there is more frequently simple diminution of secretion, as more or less all of the organic functions are in abeyance. But the most frequent variation of secretion is a disorder of its evolution. It is often normal for a short period (during the first twenty to forty minutes after the test-breakfast), and thereafter is insufficient, constituting, when the examination of the contents is made only at the end of one hour, one group of the cases with diminished activity. In another group of cases the evolution of secretion is delayed, and acid first remains free after more than an hour has passed. There is no perceptible diminution of ferment secretion, unless there be subnutrition. In adenasthenia gastrica, on the other hand, which is an independent dynamic affection, secretion of the acid, and often of the ferments, is deficient throughout the period of digestion, and there is none of the peculiar discomfort and irritable unrest. The disorders of secretion are neither persistent nor characteristic, and are in probability caused by vasomotor disturbances.

In neurasthenia gastrica the stomach usually empties itself within the normal period, and the motor function has

consequently been supposed to be normal; but this is not always a just conclusion. The stomach is often tonically, but not painfully, contracted. Swallowed air accumulates in it, and is often only got rid of by belching, the cardia yielding before the firmer pylorus. If the accumulated air does not escape, it may eventually cause anxiety, shortness of breath, palpitation, and sometimes a sense of impending death. The stomach, on physical examination, is then found contracted on the contained air, which can not be expelled by pressure; but after proper massage for a short time it escapes into the intestines, with relief of the symptoms. The most common motor disturbance, however, is simple relaxation, with splashing during the digestive period. Its independence of the quality and quantity of the food, its association with the nervous irritable weakness, the imperceptible delay in the evacuation of the stomach resulting from it, and the intolerance of remedies which do good in myasthenia gastrica, distinguish this motor disorder of neurasthenia gastrica from myasthenia, which is a distinct dynamic affection.

There are no anatomical signs of disease, and no bacteriological signs (except incidentally, as may occur in the normal stomach). It should be remembered that neurasthenia gastrica may terminate in myasthenia gastrica, and often becomes associated with chronic colitis, particularly with enteritis membranacea.

Differential Diagnosis.—Whenever the symptom-group of neurasthenia gastrica is met with in practice a most thorough examination of the whole body should be made. In this manner only can the idiopathic and secondary forms be separated. The signs of general neurasthenia should be sought; the blood should be examined for idiopathic anemia and for the amœba malariæ, and the urine for signs of chronic nephritis, of gout, and of diabetes; cholelithiasis should be excluded; the genital organs should be examined for disease, and the intestines examined particularly for chronic colitis. All these diseases may be accompanied by the digestive discomfort and uneasiness, or by the cerebral symptoms of neurasthenia gastrica. It is only by careful exclusion and by particular attention to the distinctive features of the symptoms as already described that the idiopathic affection can be defined as a morbid entity.

Some of the diseases of the stomach may be confounded with neurasthenia gastrica. Indeed, it may be a complication of most of the chronic diseases of the stomach which occur in nervous, weak, and anemic people. Practically, the

independent form of neurasthenia gastrica may have to be differentiated from chronic asthenic gastritis, ulcer, gastroptosis, atypical forms of cancer, and myasthenia gastrica.

If the clinical expression of chronic asthenic gastritis and neurasthenia gastrica be carefully studied, it will be seen that the two diseases resemble each other only when the symptoms are shorn of all distinctive features. The symptoms of this form of gastritis are digestive, in relation with the solidity of the food and proportionate to its physiological action, and the cerebral, circulatory, and distant sensory signs (including the tender points) of neurasthenia gastrica are absent. A proper diet, strictly followed, in gastritis relieves the symptoms, but the same diet is of little value in neurasthenia gastrica. The anatomical signs of chronic asthenic gastritis are of absolute differential value—large quantities of mucus in the test-meal contents, and of gastric epithelium and of leukocytes in the early morning washings of the stomach, and the diminution of the ferments. Normal secretion and the normal digestive transformation of the food, common in neurasthenia, exclude gastritis; and rapid but slight secretory variations, not due to local irritation of the mucous membrane, are in favor of the dynamic affection. Both clinical forms of chronic gastritis, after healing, may leave the nerves which supply the stomach abnormally irritable.

Ulcer of the stomach not accompanied by severe pain, hemorrhage, or vomiting may be confounded with neurasthenia gastrica. The differentiation is not always possible. The discomfort is alike digestive in both, but it is not so exclusively and invariably excited by taking food in neurasthenia. The nerve-supply is in both alike oversensitive, but the circumscribed tender points of ulcer are epigastric and dorsal; the tender neurasthenic points are present also in other parts of the body. Digestive superacidity is the rule in ulcer; it is, however, rare, and is not persistently present in neurasthenia gastrica. In the one, secretion is normal or nearly normal; in the other, secretion is normal or increased. In the one, the motor function is normal or the stomach splashes during digestion; in the other, it is normal, or signs of obstructive retention may be present. There may be symptoms of general neurasthenia, or the discomfort may be more closely related to the state of the mind and of the spirits, and these relations would be in favor of the dynamic nature of the trouble. Each individual case may present symptoms found chiefly or exclusively in the one or the other disease; but the differentiation, even in the

presence of the somewhat distinctive features of the common symptoms and signs, is a mere balancing of probabilities. The diagnosis should often be left to the subsequent evolution of the case or be inferred from the results of appropriate treatment. The continuance of the discomfort and digestive unrest, in spite of rest in bed and a strict milk diet combined with alkalies, is strongly in favor of neurasthenia.

Exceptionally, atypical or latent forms of carcinoma may present, particularly in the early period, a symptom-group somewhat like that of neurasthenia gastrica, which may also be accompanied by emaciation; but after thorough study of the case, its etiology, evolution, symptoms, and signs, doubt is rarely permissible.

Gastroptosis and neurasthenia gastrica may be readily confounded. The displacement of the stomach is easily detected on examination. The causal relation or independence of the two diseases can only be made out with probability from the history and from the results of appropriate treatment. It matters little, for when both are present both must be treated.

Neurasthenia may also be mistaken for myasthenia gastrica —both infrequently being expressed chiefly by slight digestive discomfort and by flatulency. The absence of stagnation or retention often decides at once in favor of neurasthenia gastrica. In neurasthenia gastrica the motor function may also be insufficient, but this is seldom true except in the severe cases with emaciation and loss of strength; and the state of nutrition and of the mind and spirits, and the nervous disorders are out of all proportion to the slight motor insufficiency. In neurasthenia liquids are tolerated and evacuated more readily than in myasthenia, as is made clear by the water-test. Myasthenia is also constant, is greatly influenced by the quantity of the food, and is without tender points and cerebrasthenia and circulatory disturbances. The two affections may coexist, and only a knowledge of their order of development can suggest the causal relations of the one to the other. The typical cases of the two affections bear little resemblance if the distinctive features are closely studied.

Prognosis.—Neurasthenia gastrica is not a fatal disease, but it often proves very obstinate to treatment. It creates a predisposition to the development of congestion and inflammation of other organs through the vasomotor and circulatory disorders excited by it. The resistance of the organism to invasion is notably weak.

The benefits of treatment are conditioned by the ability of the physician completely to control the patient. The common sense, desires, and will must be united in one effort to get well under the strict employment of the proper remedies. The life—mental, moral, physical, and social—must be controlled and regulated in the severe cases. The mild cases may recover under gentler restrictions. When properly rested and fed, and firmly led into optimism by the physician, the prognosis is good and the duration of the treatment is shortened.

Treatment.—The treatment of neurasthenia gastrica should be methodical and consistent. Failure is often the result of a lack of unity and plan in the employment of remedies. Nothing is more important than the combination of all means which make for the repose and strength and conservation of the nervous system. Rest alone will do little good. Isolation alone will not accomplish much. Diet alone is often of little benefit. Drugs unaided often fail even to relieve symptoms. Repose of mind and body and good digestive hygiene are of little value when combined with excitant remedies. All sources of excessive irritation should be cut off, and all excessive waste avoided. The irritable weakness can be best relieved by suitable repose, by the conservation of energy, and by enough exercise and functional activity to develop tone and strength in the individual case. Aimless management without method either does harm or no good.

In order to control the patient in every particular it is necessary to possess his confidence and the sympathetic aid of those in communication with him. Under no other circumstances can his mind and spirits be controlled and utilized by suggestive therapy to influence the abdominal sympathetic. This mental and moral guidance is all the more imperative when the affection is cerebrogastric.

The nutrition must be good and the sleep sufficient and restful before any improvement can be expected. To this end hygienic remedies and diet are most conducive.

A warm and sunny climate, proper clothing, change of scene, rest from business and other cares, electricity, massage, active exercise, and hydrotherapy have the same value as in the treatment of general neurasthenia. These physical and mechanical remedies are most beneficial when mild and soothing. The amount of excitation to be produced varies with each individual case, but the after-effect should never be exhaustion or restlessness. The right method has been found when it increases the desire for food, secures sound

sleep, and relieves the unrest. The contrary effect is a sign of too much or too prolonged excitation.

The quantity of the food is regulated by the state of nutrition, but the diet is selected by its physiological action on the sensibility of the stomach, provided there be no associated disease or complication. Rationally, a diet that has but little action on secretion would be expected to be most suitable, and this is usually the case. Milk is, consequently, in neurasthenia gastrica, an appropriate food, and the finely-divided and thoroughly cooked cereals may be combined with it. Fats, of which the best is unsalted butter, are commonly well borne, and fulfil an essential purpose in nutrition. Fish and stewed white young meats agree better than the roasted and broiled red meats. Nothing is more disastrous than a dry diet composed of meats, unless it be a mixture of acids, salads, sweets, pastry, pies, alcoholic drinks, and highly seasoned dishes. Green vegetables can be sparingly used, while ripe and not very sweet nor acid but soft and juicy fruits can often be prescribed as nutriment and as remedies against the constipation. The diet should always be sufficient to support or improve nutrition in the particular case, and other articles should be added to it as more and more excitation of the mucous membrane is likely to be beneficial. The supreme clinical control of the rationally selected diet in neurasthenia gastrica is furnished by the subjective sensations of the patient. If the discomfort is relieved, the diet is right in its action; if the unrest be increased, the diet is wrong, unless some other error is being committed. Often the proper diet is more exciting than would be rationally expected.

A valuable local remedy in many cases is the intragastric douche, employed before breakfast or at bedtime, particularly in the severe and in the cerebrogastric cases. The temperature of the water should be regulated according to the irritability of the particular case. In the obstinate cases, with great digestive discomfort, the stomach may be first douched with water, and then one-half to one pint of a 1 : 2000 solution of nitrate of silver may be allowed to flow into the empty stomach through the douche-tube, and then be immediately withdrawn. After its removal the stomach is again irrigated with plain water. The nitrate of silver douche may be used once, or at most twice, a week.

A large quantity of tonics and nervines is usually given, sometimes with benefit but often with injury. No salines, no alkalies, and no antiseptics should be employed. Iron, in its least irritant and least constipating forms, or well-diluted

arsenic may be required by anemia. The infusion of condurango seems to be the most beneficial bitter. Small doses of the bromids (sodium or strontium) often undoubtedly do good for a short time. It may be advisable or necessary to use soporifics. The constipation is best treated by massage, oil or glycerin injections, electricity, and a proper diet. The common practice of frequently irritating the stomach with so-called reconstituent drugs can not be too strongly condemned in the management of neurasthenia gastrica.

The etiological treatment should receive careful consideration. Enteritis, intestinal irritation (tape- or other worms), enteroptosis, movable kidney, anemia, disease of the genital or urinary organs, associated gastric troubles (particularly gastroptosis), and whatever has prepared the soil or occasioned the genesis and aided the evolution of the affection, should have proper attention, in keeping with a fundamental principle of therapeutics.

CHAPTER V.

MYASTHENIA GASTRICA.

Myasthenia (*μῦς*, muscle, and *ἀσθενεία*, weakness) gastrica is a dynamic affection of the stomach, characterized by diminution of the elasticity and strength of its muscular layer. The stomach does not empty itself within the normal period, and is distended more easily by its contents, on which it retracts with less than its normal force. Myasthenia is primarily and essentially an independent dynamic affection of the stomach. Pathologically, many claim for it a place among the nervous affections of the organ. The nervous system may play a part in its genesis, but essentially it is a muscular affection. The muscle is everything, and is insufficient because it is weak or degenerated.

In its course the disease may pass through all degrees of motor insufficiency, varying from a slight deviation from health to absolute gastric retention. Clinically, the evolution of the disease may be divided into two periods: the one being characterized by stagnation, the stomach slowly emptying itself completely between meals or during the night only; the other by retention, the stomach always containing food.

Etiology.—Myasthenia is the most frequent dynamic affection of the stomach, occurring regardless of sex and at all ages.

The predisposition to the disease may be hereditary, persons being born with weak muscles as with weak nerves or mucous membranes. Whole families may have it, yet it may be impossible to attribute it to a common dietetic error or mode of life or to other common pathogenic influences. The causes of myasthenia are exceedingly numerous, and it is very difficult to determine precisely its relation to heredity. The very frequent occurrence of the disease in families possessing an undeniable hereditary taint—the neurotic, the arthritic, and in families afflicted with the series of diseases characterized by slow and imperfect catabolism—would seem to indicate that a myasthenic predisposition may be inherited.

The disease is more frequently acquired, and it is rare that a myasthenic person can pose with much show of reason as a victim of heredity. A mild, and often a temporary, form is produced by mental fatigue and by depressing moral causes. The shock of fear, of accidents, or of grave misfortune may be the cause of the relaxation of the voluntary and involuntary muscular systems; and myasthenia gastrica can be thus produced, particularly if the shock comes during gastric digestion. Prolonged mental and physical fatigue can have the same result. But myasthenia gastrica produced in this acute manner is most frequently mild in degree, and recovery may be rapid; but under conditions favorable to its further development the disease may become progressive and serious.

Myasthenia gastrica is an almost constant sequel of severe acute diseases. During convalescence, when the appetite represents more closely the needs of nutrition than the functional power of the stomach, the weak organ is often overloaded, and the gastric myasthenia frequently persists in spite of the improvement in the general strength and in nutrition. Typhoid fever, influenza, and diphtheria deserve particular mention in this connection, though many other adynamic diseases leave the stomach in the same condition.

Many chronic diseases are predisposing or efficient causes. It is one of the gastric affections of the initial stage of pulmonary tuberculosis. Diseases of the heart, of the lungs, and of the liver, accompanied by congestion of the portal circulation, often cause myasthenia gastro-intestinalis. It is very common in gout, in uricemia, in biliary lithiasis, in diabetes, in severe anemias, and after severe hemorrhages. All dis-

eases accompanied by malnutrition or subnutrition are predisposing causes.

Myasthenia may be a complication of many of the diseases of the stomach, and its development renders the situation much more serious. Among the many causative gastric diseases may be mentioned chronic gastritis (chiefly hypersthenic form), displacements of the stomach, and carcinoma.

The disease may also be caused by dietetic errors, particularly intemperance in eating and drinking, the overtaxed gastric muscle becoming fatigued, insufficient, and less retractile. Very large and bulky meals, large quantities of effervescent drinks, large drafts of water in summer, rapid and excessive beer-drinking, and the drinking of large quantities of hot water or of mineral waters are common errors, which, repeated often, may directly produce myasthenia. Myasthenia, according to its severity, may be accompanied by stagnation or by retention of the gastric contents.

1. MYASTHENIA WITH STAGNATION.

Clinical Description.—Myasthenia with stagnation is very variable in its severity. The degree of stagnation measures precisely the degree of myasthenia. The period of gastric digestion (which naturally varies with the quality and the quantity of the meal) may be but slightly prolonged, or the stomach may get a short rest only in the early morning. Two forms should be clearly separated, the one being mild and the other severe. In the mild form of stagnation myasthenia the stomach is empty later than it normally should be, but the evacuation of the contents of the stomach is complete between meals. In the severe form of stagnation myasthenia the stomach is not empty before the midday nor before the evening meals, but is empty for a short period before breakfast. The inability of the stomach to evacuate its contents during the twenty-four hours characterizes the retention form of myasthenia, in which form the stomach always contains food under the circumstances imposed by ordinary dietetic customs. Both mild and severe stagnation myasthenia may be latent, the disease being expressed only by its physical signs. The stomach empties itself later than in health, remains flaccid, as evidenced by splashing, throughout the digestive period, and distends excessively under the weight of a moderate quantity of contents. The balance of nutrition is maintained, but it is impossible to increase the weight of the body. The patient

does not complain, but is exposed to digestive disorder when a very large meal is eaten, or when the usual quantity of food is taken while the body and mind are fatigued.

This latent period may last a number of weeks, or even years, before the clinical period develops. This is the common mode of development when the predisposing cause is heredity, or when the myasthenia follows acute or chronic disease, or when it is due to faulty digestive hygiene. The clinical period may appear suddenly, after shock or depressing emotions, or physical or mental fatigue, or after a dietetic error or excess.

The symptoms of the clinical period are digestive, the short intervals when the stomach is empty being passed without discomfort. Soon after food is taken the stomach feels full and heavy, and the air swallowed and the carbon dioxid set free from the carbonates of the food and the saliva by the secreted hydrochloric acid distend the stomach, often to such a degree that the clothing must be loosened. The gas is removed by frequent belching, bringing with it often a little fluid into the esophagus or mouth; and this is one of the most constant symptoms of the trouble. The distention of the stomach is thus diminished, but the sensations of weight and fullness only gradually disappear as the stomach lazily empties its contents into the duodenum. If the meal be large the gastric distress may become greater and greater, until the stomach empties itself by copious vomiting, after which there is no more trouble until another meal is eaten. The duration and the intensity of the symptoms are dependent on the quantity of food eaten, and in the mild cases the symptoms may appear only after the chief meal. In the severe form the muscular elasticity is still further diminished, and the discomfort may occur after each meal, and may, indeed, sometimes be produced by drinking a glass of water. The appetite, which is usually good before meals, may be quickly satisfied. A symptom of differential value is the relation of the gastric symptoms to the quality of the food, the symptoms being excited by both fluid and solid food, fluids often producing as much distress as an ordinary mixed meal. This symptom is exceedingly rare in any other uncomplicated disease of the stomach. Such is the simple form of stagnation myasthenia, characterized by digestive symptoms in close relation with the quantity and the fluidity of the food; by belching and regurgitation; by a mere maintenance of the balance of nutrition; by physical and mental exhaustion after moderate exercise or effort; and by the physical and functional signs of myasthenia.

But the disease does not always remain so mild and simple. The stagnation is a favorable condition for fermentation, and the regurgitations may have the odor and taste of organic acids; but we would particularly emphasize the fact that fermentation is rare even in the severe form of stagnation myasthenia. In some cases, however, it does occur irregularly and intermittently. To the symptoms already enumerated may be added gastralgia, pain, often nausea and vomiting, headache, sometimes insomnia, and mental, moral, and physical depression, vertigo, peripheral and central disorders of the circulation, erythema, urticaria, slow and labored action of the heart, with a reduplication of the second sound.

In consequence of the prolonged sojourn of the contents in the stomach (and, rarely, of fermentation), the gastric glands may become irritable, and secretion may continue for a short time after the evacuation of the chyme into the intestines, or there may be simple hyperchlorhydria. This condition is expressed by gastric pain, hydrochloric heartburn and pyrosis, thirst, and sometimes a sharp appetite and headache. In still other cases the secretion may become continuous during the day, and the uninterrupted irritation may produce the hypersthenic form of chronic gastritis, which is a most serious sequel of myasthenia.

In stagnation myasthenia the bowels are usually constipated, but when myasthenia intestinalis (a common complication or association) becomes well marked, the whole clinical aspect of the case is changed. The nervous and constitutional symptoms (attributed wrongly by some authors to gastric auto-intoxication) appear; and there is constipation, insomnia, headache, neurasthenia, oligocythemia, chlorosis, disorders of the circulation, and hepatic congestion. The intestinal myasthenia may become the most prominent trouble and be manifested by periodical colalgia, localized colitis, spasm alternating with localized dilatations, intestinal supersecretion, and the discharge of muco-albuminous membranes infected with bacteria. When the intestines become involved malnutrition begins, and may lead to extreme emaciation and intestinal cachexia.

The preservation of the strength, the weight, and the appearance of good health is a marked characteristic of simple myasthenia gastrica with stagnation. The disease itself has no influence on general nutrition, the food being eventually as well utilized as in health. If emaciation occurs, it is due to an insufficient or an improper diet or to a complication. The symptoms being digestive, and increasing in pro-

portion to the quantity of food, and the appetite, even in the mild cases, being often satisfied with a few mouthfuls, the patient is very likely to eat less than is necessary to supply the nutritive demands of the body. It is not rare, also, for a myasthenic to adopt an exclusive monotonous diet, as the consequent diminution of the quantity of food gives relief. Whenever, in myasthenia with stagnation, emaciation exists, starvation, or a gastric complication, or an associated disease of another organ will be found. The urine, both in its quantity and quality, is unchanged, except when there is an associated secretory disorder or an insufficient quantity of fluids is taken.

The objective signs are more characteristic than the subjective symptoms. These signs are conditioned by the state and the degree of efficiency of the muscular layer, which distends easily and retracts imperfectly on the contents. The stomach empties itself slowly but completely, and even in the severe stagnation form no food is found in the stomach in the morning before breakfast.

A great deal of time may be spent, and partly wasted, in the endeavor to determine with exactness the size of the stomach by percussion, by the limits of the splashing sounds, by distention with gas or air, and by electric illumination. The size of the stomach bears no relation to its motor power, but an extension of the average limits of the stomach would suggest the presence of a physiologically large stomach or of one of a group of the diseases of this organ, of which group of diseases the stagnation form of myasthenia is a member.

In myasthenia with stagnation inspection is negative—no visible peristalsis, no particular changes of the form of the abdomen, but at times the distended stomach may produce a visible prominence.

On palpation the stomach will never be found firmly contracted and resistant, but flabby and flat, unless distended with a very large quantity of contents, when its form, size, and position may be sometimes determined.

Percussion gives some signs of diagnostic value. The pyloric portion may extend beyond the normal limits to the right; but it may be impossible to demonstrate this if the hepatic flexure of the colon is filled with gas. The lower border may often, but by no means always or necessarily, be below the line joining the umbilicus and the tip of the cartilage of the ninth rib. During digestion, if the time be taken to search for the sign, the percussion note over the

uncovered triangle changes with the peristaltic movements, and it changes also over the area of the stomach accessible to percussion when the position of the patient is altered.

Myasthenia, however, can not be detected by inspection, palpation, and percussion, nor by inflation and electric illumination, for the object is not the determination of the size, form, and position of the stomach, but the estimation of the tone and strength of its muscular layer. The methods of Penzoldt and Dehio are of more value if used with the proper precautions. The liver and the stomach are first mapped out by palpation and percussion, while the patient is recumbent and fasting. The position of the left lobe of the liver and of the greater curvature of the stomach should be next determined as accurately as possible while the patient stands erect. It is necessary to do this while the patient is standing in order to make allowance for the descent of the abdominal viscera produced by the erect position. This descent varies from a fraction to two or three inches, and the greater curvature would be displaced downward, not by relaxation of its walls, but by total descent. After the new boundaries have been marked, half-glasses of water are administered after short intervals, during which the change in the location of the greater curvature is determined by percussion, the patient being erect. The distance of the dislocation is directly proportionate to the myasthenia, and the quantity of water required to produce the greatest descent is inversely proportionate to the myasthenia. Generally, a liter of water does not cause the greater curvature of the normal stomach to descend below the umbilicus. Many difficulties exist in the practice of these methods, and we rarely employ them.

The most valuable physical sign of myasthenia with stagnation is percussion splashing located in the stomach, and this alone is sufficient to establish the diagnosis of motor insufficiency when the proper precautions are taken. These splashing sounds are usually absent in the healthy, vigorous stomach at all moments of the digestive period except toward the end of the digestion of a very large meal. The myasthenic stomach splashes throughout the digestive period, and also later than the moment when the normal stomach should be empty, the evacuation of the stomach being delayed. Two glasses of water given on an empty healthy stomach are passed into the duodenum without its being possible (or, rarely, for a period of a few minutes) to elicit gastric splashing. The myasthenic stomach, under the same circumstances, splashes

for two hours, or even longer. In the mild form, and in some of the cases of the severe form of stagnation myasthenia, an examination may give intermittent splashing (during a meal or after the administration of water). The stomach has enough tone to retract during peristalsis, but it relaxes completely during the periods of peristaltic inactivity. If a glass of water be given on an empty stomach, the gliding method of palpation shows the nearly empty stomach to be flabby, non-retracted, extending over a large area and the greater curvature, caught between the compressing fingers and the posterior abdominal wall, offers little resistance to fixation during expiration.

While the splashing sounds give valuable information concerning the tone and power of the muscular wall, the stomach-tube may be used to determine the motor insufficiency with great exactness. Seven hours after a Leube-Riegel meal the stomach should contain no food. After the test-breakfast the quantity of contents, determined exactly by the methods of Mathieu or Strauss, is greater than normal, the increase above the 150 c.c. revealing the motor insufficiency. The increased quantity of contents may, however, be due to supersecretion or to swallowed saliva. The emulsion meal of Mathieu, or our absorption test-meal, would eliminate this possible source of error. The stomach before breakfast or the morning after the administration of Boas' evening test-meal, contains no food in the stagnation form.

We commonly adopt the following method of procedure, the existence of motor insufficiency having been revealed by the splashing sounds or by the excessive quantity of contents one hour after the Ewald-Boas test-breakfast. If the stomach splashes in the early morning before any food or fluid has been taken, a Boas evening meal is ordered, and the stomach is examined the next morning for retained contents. If the stomach contains no food, retention does not exist. A breakfast consisting of two soft-boiled eggs, one roll, a little butter, and a cup of weak tea or coffee (milk and sugar allowed) is ordered at 8 o'clock, and at 1 o'clock the stomach is examined. If it be found empty the severe form of stagnation does not exist. If it contains remnants of the breakfast there is severe stagnation, which may be due to myasthenia or to obstruction. Two glasses of water are given on the following morning before breakfast. One and one-half hours later the stomach is examined. If the stomach is empty there is no myasthenia, but there is obstructive stagnation. If the stomach is not empty, the myasthenia is proportionate to the

quantity of water which is found in the stomach. The quantity of water may be exactly determined by introducing 100 c.c. of a one per cent. solution of dextrose or sugar, mixing it with the stomach-contents, and subtracting 100 from the result obtained by dividing 100 by the estimated percentage of sugar in the expressed dilution. If the stomach secretes excessively, the analysis of the liquid withdrawn will reveal it. This procedure constitutes the important water-test, which not only reveals the myasthenia (a "dyspepsia of liquids"), but distinguishes myasthenic from obstructive stagnation.

In stagnation myasthenia the stomach is empty in the early morning before breakfast, but digestion and secretion are prolonged. The stomach works longer than it should and secretes longer than it should; but myasthenia does not present a constant chemical type, for secretion displays irregular variations. The three stages of digestion—rise, continuance, and decline—may be simply prolonged, with no noteworthy quantitative variations of the total hydrochloric acid from the normal type. More frequently free HCl appears abnormally early and the combined HCl gradually increases, and during the latter stage of digestion is abnormally high, for the secreted HCl is not rapidly utilized and the digestive products accumulate. The period of decline of digestion is prolonged like the other stages, and it does not end suddenly, as often happens in obstructive stagnation. The lines representing the evolution of the free HCl and of the combined HCl may be irregular, with sudden rises and falls, for the evacuation of the contents may be irregular and in spurts; but these lines may also be irregular in obstructive stagnation. In stagnation myasthenia the total HCl (H + C) may be less than normal, the combined HCl gradually increasing as digestion proceeds. If the HCl secreted is greatly and constantly diminished, the co-existence of stagnation should arouse suspicion, for this combination of functional signs is rare in myasthenia; but it is frequent in carcinoma, in chronic gastritis with infiltration of the muscular layer, and in the complicated cases of pyloric obstruction. In adenasthenia gastrica and in simple chronic asthenic gastritis the stomach empties itself with normal or abnormal rapidity, for its motor power is good and the digestive work which it is capable of doing is quickly finished. To detect these abnormalities of the evolution of secretion it is necessary to remove and analyze the contents of the stomach thirty, sixty, and one hundred and twenty minutes after the beginning of the test-breakfast. With the exception of these evolution disorders

there are no abnormal secretory signs in stagnation myasthenia. We would particularly emphasize the almost invariable absence of abnormal bacteriological signs. There is no excessive germ growth, no fermentation, and no gas formation in the fermentation tube tests. Very rarely there are a few yeast cells.

The evolution of myasthenia with stagnation is variable. Developing as the sequel of an acute disease, it may rapidly disappear under proper treatment. It may be arrested at any stage, whatever be the cause. When a hereditary predisposition exists it may last from childhood to old age, but be controllable by proper digestive hygiene. However caused, the form with stagnation may rapidly or slowly grow into the form or period characterized by retention. Its course may be broken by latent periods, characterized only by physical signs, or by exacerbations due to physical, mental, or nervous exhaustion, or to dietetic excesses.

Diagnosis.—The diagnosis of myasthenia gastrica is easy if the modern methods of examination be employed. The restriction of the symptoms to the prolonged digestive period and their relation with the quantity and fluidity of the food; the delayed evacuation of the flabby, easily distensible, and slightly retractile splashing stomach; the absence of food in the fasting morning stomach; the absence of a characteristic germ growth or secretory deviation; the mode of development and the usual maintenance of nutrition—are sufficiently characteristic.

Differential Diagnosis.—The diseases most likely to be confounded with the stagnation form of myasthenia gastrica are neurasthenia gastrica, displacements of the stomach, pyloric obstruction, cancer, chronic gastritis, and myasthenic gastric retention.

In both neurasthenia and myasthenia the subjective symptoms are digestive, and may be similar; but in neurasthenia the subjective symptoms are not proportionate to the quantity of the food, and are commonly more pronounced with solids than with fluids. In myasthenia the symptoms are greatest after large meals, and are least after small meals of finely-divided solid food with little fluid. Gastric excitants, particularly condiments and irritant drugs, increase greatly the uneasiness and discomfort of neurasthenia, and are comparatively well borne in the mild form of myasthenia. In neurasthenia the abdominal plexuses are sensitive, and pressure, particularly during the period of functional activity of the digestive tube, over the four points situated a little below

the ensiform process, to either side of the umbilicus, and above the symphysis pubis, produces an indefinable discomfort. Care should be taken to exclude a tender left lobe of the liver, a filled or an inflated colon, and a distended bladder, tenderness over which may be mistaken for sensitive sympathetic ganglia. With these may be found other signs or symptoms of neurasthenia, as neuralgia, spinal points, headache, and insomnia; but none of the signs enumerated are conclusive, as neurasthenia may generate or may be associated with myasthenia. The functional signs, however, are distinctive. In neurasthenia gastrica both the mechanical and the chemical functions of the stomach are normal, and consequently the presence of gastric splashing under the conditions already given, and the evidences of constant motor insufficiency furnished by the tube and by test-meals, exclude simple neurasthenia gastrica with certainty.

The vertical displacement and the prolapse (gastroptosis) of the stomach may be confounded with myasthenia. The dislocations of the stomach may be symptomless until some complication develops, and the most frequent complications are myasthenia and motor insufficiency, due to partial obstruction. In vertical dislocation the pyloric portion is displaced downward and to the left; the percussion area of the stomach does not cross the median line, and remains high, as in health. In gastroptosis the lesser curvature descends, and other abdominal viscera are usually also displaced. In both forms of displacement the stomach may splash. The inflation of the stomach and the other physical methods of examination usually reveal clearly the position of the stomach. It should not be forgotten that the displaced stomach is often myasthenic. The abdominal belt and the water-test, and possibly visible peristalsis and a palpable, firmly contracted stomach, furnish the only means of distinguishing myasthenic stagnation from the obstructive stagnation produced by displacements of the stomach or by enlargements of the liver.

The differentiation of myasthenia from pyloric obstruction may be difficult. In making this distinction the clinical history may be very valuable when it gives a group of symptoms characteristic of a disease which is likely to produce an obstructing deformity. The most common diseases of the stomach which produce obstruction are ulcer and carcinoma. If there be a history of gastric hemorrhage, of epigastric pain bearing the character of that of ulcer, and other gastric symptoms in the characteristic relation to the ingestion and

the qualities of the food, it is very probable that the motor insufficiency is due to obstruction. The motor insufficiency of the stomach may be due to other diseases than that of the stomach itself, such as duodenal stenosis, perigastric and duodenal adhesions, the pressure of tumors and of displaced organs. The obstructed stomach contracts on its contents and retracts when it is empty. In marked contrast is the weak, easily distensible, slightly retractile stomach of myasthenia. During the performance of lavage in myasthenia the water flows in rapidly and continuously, a suction whirlpool may be produced by holding the funnel high, and the outflow is slow and steady; but in obstruction the inflow is slow and may temporarily stop, and the outflow is strong and often in spurts. In myasthenia expression is difficult and always incomplete; but in obstruction the greater portion of the contents can be easily removed by expression. The muscular layer of the obstructed stomach, in the effort to establish compensation, becomes hypertrophied, stronger than normal, and the moderately filled organ may be felt by the educated fingers rounded, sharply limited, resistant, alternately relaxing and contracting. The method of Dehio shows no increase of normal distensibility. Splashing, if it exist, is limited to a small area during the digestion of a meal. After a glass of water taken on an empty stomach, splashing is often difficult to produce, is limited to the area of the uncovered triangle, and the gliding method of Glénard is impracticable, or reveals a firm stomach within the normal boundaries and with walls brought in contact with difficulty. In favorable cases with thin and relaxed abdominal walls, peristalsis, and sometimes antiperistalsis, may be visible during the digestive period. Unless the stomach is displaced or is physiologically large, it remains within its normal boundaries, both when full and when empty. The healthy and physiologically large stomach secretes, digests, and empties itself in a normal manner. [For a full discussion of the differentiation of obstructive stagnation and myasthenic stagnation see the article on obstruction of the pylorus, where the differential value of the water-test is also made clear.]

Motor insufficiency is one of the earliest and most constant signs of carcinoma of the stomach. It may be due to pyloric obstruction, to infiltration of the muscular layer, to edema and malnutrition from obstruction of the venous and lymphatic circulation, and, probably, also to reflex nervous influences. Myasthenia is very slow, often stationary, in its course, occurs at all ages, is amenable to treatment, when

simple is without deleterious influence on nutrition, and presents a normal series of secretory and bacteriological signs in marked contrast with those of carcinoma. Cancer is rapidly progressive; is most frequent after forty; is only temporarily influenced by treatment; causes progressive emaciation and loss of strength, with blood and urine changes; may be accompanied by transient edema of the lower extremities; by enlargement of the inguinal and supraclavicular glands, by secondary nodules in the liver, and by a palpable tumor. If the cancer involves the stomach, the chemical and bacteriological signs are distinctive; but these are absent if the motor insufficiency is due to compression by cancer of a neighboring organ, such as the pancreas or the gall-bladder. In cancer, particularly in the early and obscure stage, the stomach is neither flabby nor easily distensible as in myasthenia.

Chronic asthenic gastritis is rarely accompanied, except in the advanced stage of some cases, by motor insufficiency. Consequently, none of the physical and functional signs of myasthenia exist in this disease. The stomach is normal in size; there is no abnormal splashing; the hydrochloric secretion and the ferments are constantly diminished; there is always an excess of mucus; after a test-breakfast the contents are thick and contain little fluid, and two glasses of water are evacuated from the stomach within two hours. Cases of chronic asthenic gastritis which have become complicated by motor insufficiency do not differ materially from myasthenia which has become complicated by asthenic gastritis, except in their etiology and evolution.

A stomach of any size may become myasthenic, but a large stomach will not be confounded with a myasthenic stomach, except when the diagnosis is based on the position of the greater curvature, a mistake which is sometimes made by the inexperienced. Myasthenic stagnation is easily distinguished from both myasthenic and obstructive retention by the presence of food in the stomach before breakfast after the Boas evening meal and by the active fermentation which exists in retention.

Treatment.—The prophylaxis of myasthenia is very important. During convalescence from acute diseases, and during the course of exhausting diseases, the diet should always be regulated so as to favor the weak gastric muscle, and means should be adopted to preserve and to revive its power. If there be a hereditary predisposition, digestive hygiene should be regulated so as to require no extraordinary

mechanical work of the stomach, and the neuromuscular system should be developed by a properly regulated life. The same prophylactic measures are an essential part of the treatment of all diseases of the stomach likely in their course to become complicated by myasthenia, the development of which marks a danger-point in their evolution and often necessitates a complete change of the treatment.

The treatment of the established disease is simple, and the object is clearly defined by its pathology. The weak muscle must be favored in its work, its strength must be developed, and enough food should be taken to keep the body well nourished.

A proper diet is consequently a matter of the first importance. The food should be nutritious, in small bulk, and indifferent in its action on the stomach. Some authorities claim that a dry diet is essential and best, and recommend frequently repeated meals; but in our experience a glass of fluid is an advantage, and the stomach regains its tone more rapidly when allowed as long an interval of rest as possible. In the severe form it is not advisable to give frequent meals; the stomach should be given time to empty itself before introducing more food into it. An increased number of meals necessitates an approximation of the meals, and this disadvantage may outweigh the benefit derived from the diminution of the quantity of each meal. The one or the other method may suit a particular case best, and it may be wise to control the plan of feeding adopted by daily examinations and by the frequent use of the stomach-tube. It is best, however, when the food is badly utilized by the intestines, or when the stomach is capable of managing only a small quantity, to divide the total quantity of food required for the twenty-four hours into four equal parts, to be given five hours apart. Overtaxing the stomach is thus avoided and an interval of repose is secured between the meals.

An excess of sweets and fats is injurious in myasthenia. The one increases the quantity of secretion and is liable, on account of the stagnation, to induce fermentation; and the other delays the evacuation of the stomach. But fat in the form of fresh butter should be given in sufficient quantity to meet the requirements of nutrition, and for theoretical reasons is too often forbidden, for experience shows that it may be well borne, and this rich force-producing and nerve-building food is essential in moderate quantity. It is customary to exclude sweets, but the exclusion of this valuable class of foods is also an error, for it is only necessary to limit

the quantity and to select the most digestible of the foods which contain sugar. The fear of fermentation in stagnation myasthenia is not well founded, and it is rarely necessary to do more than to select the fats and sweets and to forbid their excessive consumption.

The diet should not be exclusive, but mixed, and should consist of a combination of finely-divided, tender, and lean meats; fish; the more nutritious and easily digestible cereals, such as rice and the preparations of wheat and hominy or cornmeal mush, thoroughly cooked; a small quantity of vegetables which do not possess a great deal of waste matter; fresh butter; and some sweets. Preparations of eggs may also be allowed, but too much of the yolk will not be well borne. An exclusive or chiefly milk diet is often prescribed, but is an experiment, and, on account of the large quantity required to support nutrition, is very objectionable; but milk, when well borne, may be permitted as a drink. A cup of coffee once or twice a day may not be injurious. Tea, cocoa, and chocolate should be prohibited, but Hauswaldt's "Vigor Chocolate" may be tried. A small quantity of alcohol, if the patient is accustomed to its use, may be permitted at meal-time, and will usually promote both absorption and the evacuation of the stomach. A little pure old whisky or brandy may be taken, well diluted, or a light wine may be permitted; but often no drink will be found to agree better than a glass of plain water, just cold enough to be refreshing. A small quantity of ripe, seedless fruit should not be injurious.

This is the diet of simple myasthenia with stagnation: Small in bulk and nutritious; finely divided; mixed; very little fruit and vegetables; a moderate quantity of sweets and fats; nitrogenous foods and cereals as freely as in health; and a limited quantity of fluid. We are not advocates of a dry diet in myasthenia, and at least three pints of fluid should be taken in the twenty-four hours. The gastric splashing has led logically to the recommendation of a dry diet. This we believe to be a mistake, and consequently permit a glass of fluid (water, coffee, meat broths, possibly milk, or "Vigor Chocolate") with each meal. Two or three more glasses of fluid must be given during the twenty-four hours, at such times as will interfere the least with digestion, or it may be advisable to give part of the required fluid by rectum.

In the erect position the contents of the myasthenic stomach weigh down and distend the pyloric portion, and may produce an incomplete obstruction of the duodenum at its

point of attachment to the vertebral column. It is not the liquid alone, but the weight of the total gastric contents and the erect position which add to the labors of the weak muscle. The myasthenic who overloads his stomach and goes to work or exercise immediately after meals is not likely to do well. If the mechanical work required of the stomach in a particular case is greater than it can perform, the insufficiency will increase and retention will result. One or two hours' rest after meals is wise and beneficial.

Where the myasthenia is complicated, the chemical and bacteriological signs will require consideration in selecting the diet. These complications or accidental associations are fermentation, excessive secretion (adenohypersthenia), gastritis, and intestinal diseases. It is still, however, the functional power of the stomach and intestines and the physiological action of the food which decide the diet after the possible excessive or peculiar germ growth is arrested. A diet so regulated as to support nutrition and to diminish the mechanical work of the stomach places the organ in a favorable condition for regaining its lost muscular power. But other remedies may also be employed to strengthen the weak muscle. The most important of these are exercise, massage, electricity, hydrotherapy, and a few drugs.

The principle which should regulate the strength and dose of these remedies may be the contrary of that which usually guides practice. The greater the myasthenia, the milder and less excitant, in some cases, should be the remedy. The weak gastro-intestinal muscle may be excessively irritable, and overexcitation or stimulation does harm. This injurious action is clearly demonstrated by the effect of a strong purgative in well-marked myasthenia of the colon, producing colic and localized contractions persisting for hours. No remedy which strains or exhausts a weak muscle can increase its strength. The dose of the muscular excitant must fall short of producing irritable spasm and exhaustion.

The exercise, which is best taken in the open air, should not begin earlier than one hour after meals, and should always be moderate, so that the slight sense of fatigue disappears after a few minutes' rest.

Abdominal massage is a remedy of value. If the stagnation is pronounced, it may be employed late in the evening—four hours after meals—to empty the stomach, the organ being left contracted under the influences of the skin reflexes excited by circular movements of the tips of the fingers around the umbilicus and over the stomach. It is often advisable to

massage the abdominal muscles, but the intestines should be left alone to digest and to absorb their contents during the night. The general abdominal massage, if indicated by intestinal stagnation, should be performed in the morning, fasting or after the administration of a glass of water. The best methods of using water in the treatment of myasthenia are the intragastric spray and the Scottish douche, in the manner recommended to tone the abdominal sympathetic. If the patient tolerates the tube, the intragastric douche with plain water will be found valuable, particularly in the cases where suggestion is likely to prove a remedy of value. The tonic influence of a shower-bath is thus brought directly to the gastric wall. The intragastric spray may be employed twice a week, in the morning before breakfast, care being taken to avoid overloading the stomach with water and to leave the organ empty. There is in the stagnation form of myasthenia no indication for stomach washing.

Electricity may also be often employed with advantage, strong currents of low density being most beneficial. Intragastric faradism and cathodal galvanism are the best methods, and minute directions for using electricity to tone the stomach will be found in the section on General Medication.

A few drugs are of great value in the treatment of myasthenia. Of these, strychnin deservedly holds the first place. Ergot is also valuable when well borne. Hydrastinin muriate is often beneficial. Quinin (alkaloid preferable to its salts) is almost as valuable as strychnin. The prolonged use of ipecac in very small doses is also beneficial. Two or more of these drugs may be given combined. The other medicines are those commonly used to control gross symptoms.

Constipation may be a troublesome association, but is usually relieved by a proper diet, by massage, by electricity, by gluten suppositories, by glycerin suppositories, and by the general hygienic remedies directed against the gastric myasthenia. An occasional injection of water, or water and a teaspoonful of glycerin, may be required. Cascara sagrada in barely efficient doses (combined with hyoscyamus or belladonna) may be prescribed. A pinch of Turkish rhubarb after the evening meal may be all that is needed. But the time required for the patient to get well is prolonged by the use of purgatives, and no good is done the myasthenic stomach by the frequent employment of laxatives.

2. Myasthenia with Retention.

Stagnation myasthenia may remain stationary for a long period, but its inherent tendency is to increase. Retention myasthenia is, consequently, the natural sequence of the stagnation form. The prolongation of digestion, which necessarily results from stagnation myasthenia, increases the mechanical work which the stomach has to perform and requires abnormal endurance of its weak muscle. The normal stomach would hypertrophy under the increased work demanded by the prolongation of digestion and of secretion, but, unfortunately, the myasthenic stomach does not; its task must be lightened before the weak muscle will begin to recuperate. Furthermore, the myasthenic stomach creates new difficulties for itself. Normally, the pyloric end of the stomach, as it becomes distended by the contents of the stomach forced into it by peristalsis, extends downward and to the right, and is elevated by rotation forward and upward, thus relaxing the gastrohepatic ligament and making it easy for fluids to pass. In myasthenia the enlarged pyloric antrum simply descends in the abdomen, tightens the gastrohepatic ligament, and produces an angular constriction, which renders the evacuation of the stomach more difficult. If the patient be emaciated, or if abdominal tension is low, the production of the duodenopyloric constriction is facilitated. In spite of this difficulty, and in spite of the increase of mechanical work, the stagnation myasthenia may persist for a long time without progressing to a severer stage, or retention may eventually result. The advent of retention is favored by a very large meal, by excesses, by fatigue, by enlargement of the liver, and by acute or chronic exhausting diseases.

Clinical Description.—When the myasthenia becomes so great that the stomach never completely evacuates its contents into the duodenum, the symptomatology changes. To the digestive symptoms proper to myasthenia are added those due to the retention and the fermentation.

The appetite is usually poor, and decreases with the activity of fermentation. Thirst may become intense, but is in direct relation with the system's loss of fluid by retention and by vomiting. The patient, after a restless night, awakes in the morning, commonly with a headache, depressed and tired. Immediately after meals the stomach feels heavy and full and the abdomen becomes flatulent and distended. The flatulency increases with the movements of the digestive tube, and

large quantities of gas are removed by repeated belching, often bringing a small quantity of a sour and bitter fluid into the mouth. There is constant nausea, and sometimes vertigo, if there is butyric fermentation or putrefaction. The products of fermentation produce sometimes a burning, local pain, and if active fermentation continues in the intestines it produces also colic, and, rarely, diarrheal movements; but obstinate constipation is the rule in myasthenia with retention. During the digestive period the headache and nervous symptoms increase. The symptoms diminish after a number of hours, but there is no interval of gastric repose or comfort. From meal to meal the retained contents, in active fermentation, accumulate in the stomach, until about every second or fourth day the overloaded organ is relieved by copious, effortless vomiting, occurring often without nausea. The system is thus robbed of its nourishment by vomiting, and by fermentation beginning in the stomach and extending to the intestines, and is also slowly poisoned by germ products.

Vomiting, however, is not so common a symptom of myasthenic retention as of that due to pyloric obstruction, and is usually incomplete, less copious, and more apt to occur three or four hours after a meal than at night. The vomit consists of a brownish mixture of a bad odor, like that of "dead" yeast fermentation, very acid, often bitter, containing mucus and sometimes blood and bile, and separates, on standing, into three distinct layers, the upper being fatty and foamy, the middle fluid and cloudy, and the lower consisting of solid residue of food mixed with germs and giving off bubbles of gas, often carrying with them on their way to the top layer little particles of food. The vomiting becomes less and less frequent as the myasthenia increases, but when it occurs temporary relief follows.

Emaciation is a constant symptom of myasthenia with retention, and is due partly to enforced inanition. The intestines can not compensate the inefficient stomach, because the fermenting mass, in part delivered to it, is neither digestible nor nutritious, and disorders the functions of the intestines. Gastric absorption decreases with the overdistention of the stomach, and many of the so-called auto-intoxication symptoms are due to the deficient supply of food and water.

The functions of the intestines are always disordered in myasthenic retention. If the vomiting is complete it is often followed by improvement of the intestinal fermentation and of flatulency; but the constipation continues obstinate and the movements are rare, dry, and scybalous. In some cases

the stomach, irritated by the products of active fermentation, empties its contents into the intestines and produces a fermentative diarrhea. Often the intestines are also myasthenic, and, as a consequence, enteritis membranacea may eventually develop.

The urine is always diminished in quantity in proportion to the insufficient absorption and to the loss of water, and it is of high specific gravity. Consequently, the quantity of urine passed in the twenty-four hours may be taken as a rough measure of the degree of retention, notice being taken of the quantities of water ingested and vomited or, rarely, eliminated by diarrhea. The chlorids are diminished and the phosphates are increased and precipitated in large quantities on heating. If there be excessive secretion, the urine may present all the characteristics found in hypersthenic gastritis. Rapid or severe emaciation may be accompanied by acetonuria.

A large number of general symptoms and diseases of other organs results directly and indirectly from gastric retention. Some find a convenient explanation of these in reflex action; others attribute them to the subnutrition, the deficiency of water, the disordered metabolism, and the diminished power of resistance of the organism; and still others, adopting the theory of Bouchard, explain their genesis by auto-intoxication. The germ products absorbed from the digestive tube doubtless play an important pathogenic part, and produce directly many of the general symptoms, but it can not be shown that more than a part of these products are formed in the stomach.

The list of these secondary troubles is a very long one. The liver is frequently periodically congested. There may also be attacks of icterus without alteration of the color of the stools. Palpitation, arrhythmia, tachycardia, reduplication of the second sound, are common heart disorders, the action of this organ (right ventricle) being also at times mechanically interfered with by the presence of the distended stomach and by contraction of the pulmonary arterioles. Bronchitis occurs as frequently as inflammation of other mucous membranes. Urticaria, acne, pityriasis versicolor, erythema, and eczema are some of the skin affections associated with myasthenic retention. The nervous symptoms are very numerous —disorders of the special senses and of speech, affections of memory, hypochondriasis, tetany, muscular cramps; attacks beginning with restlessness, jactitation, slight general convulsions, and accompanied by dyspnea and somnolence, ending in

coma. Some claim rickets to be the result, while Bouchard describes an enlargement of the second phalangeal joints as a nutritive disorder which exists only in association with gastric retention. That auto-intoxication may be the possible cause of these numerous troubles may be admitted; but a possible explanation is not necessarily always the true one, and it is certain that they have not been proven to result from gastric retention.

The Objective Signs.—The signs furnished by the examination are those of myasthenia, of retention, and of fermentation. The signs of myasthenia have been given in the description of the stagnation form or period of the disease. The second clinical form or period is characterized by the signs of retention and of fermentation.

The patient is emaciated, the skin is dry and rough, and the secretions are thickened; the extremities are often cold, and the fingers may show the nodosities of Bouchard.

The physical examination usually reveals a flabby stomach of large size. If the organ is filled, the epigastrium and the left hypochondrium are prominent, and the greater curvature may often be seen and felt running lower and to the right of the normal limits, splashing always under the same conditions as in myasthenia with stagnation. A normal size of the stomach does not exclude either form of the disease. Inflation may be employed to locate and to define more accurately the boundaries of the stomach, revealing or excluding a displacement. An important inspection sign is self-inflation of the stomach, when the viscus may be seen slowly to distend with gas and somewhat abruptly to collapse with the escape of gas through the pylorus. Peristalsis is visible only when there is obstruction to the evacuation of the stomach, and is not a sign of myasthenia. On placing the stethoscope over the stomach, intragastric bubbling can often be heard. Both the self-inflation and the crackles are signs of gas-forming fermentation, and when present in the morning before breakfast are pathognomonic of retention and of fermentation.

Another physical sign of retention is gastric splashing in the morning before anything has been taken into the stomach. This sign is never absent in myasthenic retention, but is also found in continuous secretion.

The pathognomonic sign of gastric retention is the presence of a noteworthy quantity of food in the stomach in the morning after a test-supper (Boas). To measure the degree of motor insufficiency the stomach should be washed out before the supper is eaten. The quantity of food in the stomach on the

following morning is, then, proportionate to the motor insufficiency.

The contents of the stomach, removed by vomiting or by the tube, present physical, chemical, and bacteriological properties characteristic of retention. The odor is sour, dead, often rancid. The total acidity is always high, consisting of the free and the combined acids of secretion and fermentation. On standing, it separates into three layers, with visible gas formation. The gases consist of those of swallowed air, of fermentation, and of putrefaction, including sometimes H and H_2S. Sarcinæ, yeast, and numerous bacteria are found. If the stomach is washed out and a test-breakfast given, we may obtain the secretory and digestive signs of hyperchylia or of a complicating gastritis, plus fermentation. Lactic acid is frequently found in the contents where no free HCl is present; but lactic acid is not formed under the conditions of the Boas cancer test. Gas formation is active in the fermentation tubes filled with the unfiltered and sweetened contents of the stomach.

Diagnosis.—The objective signs of gastric retention are so characteristic that the diagnosis of the condition is easy. It is often confounded with the stagnation form of the disease, both possessing in common the signs of myasthenia; but on account of the different treatment and prognosis it is practically important that the two forms should be sharply separated. The one is benign, local, unaccompanied by emaciation or auto-intoxication, the stomach always succeeding in completely emptying itself and in delivering to the body the required quantity of nutriment of all sorts. The other is a starving and poisoning disease, accompanied by emaciation, auto-intoxication, morning retention, and active gastric fermentation, the stomach withholding the amount of both food and water necessary to maintain nutrition. The physical, functional, and bacteriological signs render it possible to determine accurately where the one begins and the other ends. But gastric retention is not synonymous with myasthenic retention; consequently, myasthenic retention must be distinguished from the other diseases which are accompanied by gastric retention.

Differential Diagnosis.—Motor insufficiency of the stomach may be due to increased resistance to the evacuation of the stomach, to diminution of the evacuating power of the stomach, or to an abnormal amount of work to be accomplished. Increased resistance to evacuation is the result of obstruction. Diminution of the evacuating power may be

produced by myasthenia, by cancerous or inflammatory infiltration of the muscular layer of the stomach, and by gastroplegia. The amount of work may be made abnormal by the eating of a very large meal.

Now, gastroplegia occurs suddenly, and is usually the result of severe shock or of peritonitis. It is hardly possible to mistake this rare disease for retention myasthenia. Acute retention may be produced by a very large meal, but the stomach recovers its motor sufficiency in a few days after the large meal is evacuated into the duodenum or is expelled by vomiting. Very rarely death may occur, if the greatly distended stomach can not evacuate the semi-solid contents, the whole mass acting as one body. Acute retention produced in this manner is not likely to be confounded with myasthenic retention.

Gastric retention due to myasthenia is, then, to be distinguished from retention due to obstruction, and to cancerous or inflammatory infiltration of the muscular layer. In only one other disease of the stomach is a noteworthy quantity of contents found persistently in the stomach in the morning before breakfast, and this disease is hypersthenic gastritis with continuous secretion, which may be accompanied by pyloric obstruction, or by myasthenia, or by myositis, while the motor insufficiency may be so great as to produce retention, or sometimes only stagnation.

It may be very easy to exclude hypersthenic gastritis with continuous secretion. This usually painful disease has many characteristic symptoms, as may be seen by reading its clinical description. Anatomical signs may be found in the contents of the stomach in this disease, such as blood, a large quantity of mucus, nuclei of white corpuscles, and sometimes pieces of inflamed mucous membrane. To distinguish it from myasthenic retention it is only necessary to establish the morbid continuity of secretion, for myasthenia with secondary hypersthenic gastritis and continuous secretion does not differ from hypersthenic gastritis accompanied by myasthenia and continuous secretion except in their preceding history and evolution. Now, in continuous secretion the stomach may or may not retain food. If it does not retain food (coarse, finely divided, or in digestive solution), a noteworthy quantity of digestive fluid (more than twenty c.c.) in the stomach before breakfast makes it clear that secretion is continuous. If it does retain food, it is necessary to wash out the stomach most thoroughly in the evening. It may be very difficult to cleanse the stomach thoroughly, and it is some-

times wise to wash out the stomach in the evening and twice on the following day, during which time the patient is nourished exclusively by rectum, water only being permitted by mouth. On the following morning before breakfast the contents of the stomach are removed. If there is continuous secretion, the stomach will contain more than twenty c.c. of gastric juice. The total contents of the stomach should be estimated by the acidity method of Mathieu or by using the one per cent. solution of sugar. The properties of the morning contents may sometimes make the differentiation clear without the employment of lavage, as stated in the following propositions:

1. If lavage has not been practised after dinner on the preceding evening, and if the morning contents contain no visible remnants of food, nor deposit a flocculent sediment which the microscope shows to be composed of particles of food, nor give on analysis a noteworthy quantity of combined HCl,—there is no myasthenic retention, nor food retention from any cause; but if the stomach contains more than twenty c.c. of a digestive fluid there is continuous secretion.

2. The morning contents in uncomplicated continuous secretion are never more acid than the contents after the test-breakfast. If the acidity (H + C) of the morning contents is greater than that of the digestive contents there is retention.

3. If the lines which represent the quantity of free (H) and of combined (C) hydrochloric acid during the evolution of the digestion of the test-breakfast undergo sudden rises and falls, there is motor insufficiency, for the rises and falls are due to the irregular evacuation of the contents (obstruction) or to the infrequency of evacuation efforts (myasthenia). If continuous secretion is not associated with motor insufficiency during the period of decline of the digestion of the test-breakfast, the liquid contents are excessive, and contain free HCl and chlorids, but very little combined HCl.

4. Whenever, in the ordinary course of events and without previous evening lavage, the stomach contains in the morning before breakfast a very large quantity of secretion but no coarse particles of food, the continuous secretion is accompanied by motor insufficiency, due to weakness or disease of the muscular layer, unless the examination has been made during a "secretory crisis."

5. In continuous secretion without food retention, the morning accumulated secretion does not produce gas formation when tested (unfiltered and sweetened) in the fermentation tubes.

When continuous secretion exists—and its existence is proven by the continuance of gastric secretion after the stomach has been completely relieved of all food by lavage, aided, if need be, by fasting—to such a degree of activity as to permit the accumulation of a pathological quantity of gastric juice in the stomach before breakfast, there may or may not be food retention. If there is retention of food, the disease can not be simple myasthenia with retention, and the diagnostic problem which remains for solution is the detection of the cause of the retention which is associated with the continuous secretion of hypersthenic gastritis or of paroxysmal hyperchylia gastrica.

If continuous secretion does not exist, the retention of food and digestive products may be due to myasthenia, to obstruction, or to secondary disease (inflammation, cancerous degeneration, atrophy) of the muscular layer. Consequently, the myasthenic retention must be distinguished from obstructive retention, from carcinomatous retention, and from the motor insufficiency which complicates and results, very infrequently, from chronic gastritis.

Myasthenic retention is comparatively rare; obstructive retention is frequent. A history of ulcer, or of toxic gastritis, or of gall-stones is in favor of obstruction. A history of myasthenic stagnation, or of an acute or chronic exhausting disease, and the coexistence of phthisis are in favor of myasthenic retention. The presence of a pyloric tumor removes all doubt of the cause of the retention.

The evolution of myasthenia is ordinarily slow, the symptoms are comparatively mild in character, and the quantity of food retained is proportionate to the duration of the disease of the stomach. Obstructive retention may develop rapidly, is ordinarily a very painful disease, and the quantity of food retained is proportionate to the degree and not to the age of the obstruction.

If there is gastroptosis, the retention may be obstructive or myasthenic. If the retention is due to traction on the duodenum, support of the stomach by an abdominal belt and rest in the horizontal position during digestion will relieve the retention. Displacement of the stomach is much more frequent in pyloric obstruction (neoplasms) than it is in myasthenia. In pyloric obstruction the displacement of the stomach begins with the pyloric end, and the fundus of the stomach, as a rule, does not descend. In gastroptosis the whole stomach descends in the abdomen, and the lesser curvature and the upper border of the fundus are displaced down-

ward in proportion to the gastroptosis. The obstructed stomach retains its general form, which is lost in myasthenia.

The obstructed stomach is enlarged and hypertrophied; reflex peristalsis and contraction are strong and are easily excited; expression of its contents presents no difficulty, but it is very hard to remove all the coarse particles of food from the stomach by lavage. In myasthenic retention the stomach may be large or small; its wall is thin and flabby; reflex peristalsis can not be excited and is never visible; contraction is weak; expression of its contents is so difficult that siphonage or suction must usually be employed to obtain the contents for analysis; and after prolonged lavage it is hardly possible to get the stomach clean and empty.

In myasthenic retention there are no peristaltic pains and no cramps, and vomiting is infrequent (overflow), copious, liquid, and incomplete. In obstructive retention, peristaltic pains and cramps are ordinarily severe, vomiting may be frequent, retching, and complete, and the vomit is thick and contains plenty of coarse food, and sometimes blood, nuclei of white corpuscles, much mucus, and possibly pieces of a tumor or of the inflamed mucous membrane may be found in the vomit, in the expressed contents, or in the wash-water.

In myasthenic retention the empty stomach does not retract, and emaciation is usually not rapid nor very great so long as the stomach can furnish the system with the required quantity of water. In obstruction the empty stomach retracts, and emaciation may develop very rapidly. The enlarged, hypertrophied, and obstructed stomach may not completely retract when it is empty, and the administration of half a glass of water may make it possible to produce splashing over a very small area. The splashing can not be produced when the stomach is full, for it is then firmly contracted on its contents, except during the short intervals of peristaltic repose. In myasthenic retention the splashing sounds may be produced at all hours, day and night, and, as the rule, after most painstaking efforts to empty the stomach completely with the tube.

The evolution of the digestion of the test-breakfast is different in the two forms of retention. The test-breakfast should be preceded by thorough lavage, and the stomach should be left as nearly empty as possible. In myasthenia the first two stages of digestion may be prolonged or free HCl may appear earlier than in health, but the quantity of combined HCl gradually accumulates, and at the end of two hours the stomach contains about as much, and sometimes

more, fluid than was taken with the test-meal. Some of the contents are evacuated, but the slowly evacuated fluid is replaced by secretion during the first two hours. In obstruction the first two stages of digestion may be nearly normal, but free HCl is excessive usually after the first hour, and at the end of two hours the stomach contains a thick mixture of coarse and fine particles of bread, and usually an excessive quantity of physiological hydrochloric acid (H + C). Obstructive retention is a retention preëminently of solids, and myasthenic retention is a retention preëminently of liquids. Consequently, if two glasses of water are given on an empty stomach in myasthenic retention, the greater part of the water will still be in the stomach after the lapse of two hours, but in obstructive retention the stomach will be empty. For the same reason, in obstructive retention there is much coarse food in the early morning contents, but in myasthenic retention there is some food and a comparatively large quantity of fluid. In myasthenic retention a moderately dry diet is best borne; in obstructive retention the diet should be fluid.

In myasthenic retention physical and mental fatigue increase the motor insufficiency but have no effect on obstructive retention. Myasthenic retention may be relieved by favoring and toning the stomach muscle. Obstructive retention is persistent without surgical intervention.

The differentiation of myasthenic retention from retention due to carcinoma and to advanced chronic gastritis will present no difficulty when the characteristic signs and symptoms of these disorders are kept in memory. The persistent functional, bacteriological, and anatomical signs of carcinoma and of the terminal period of chronic gastritis exclude readily the retention due to myasthenia; but chronic gastritis and myasthenic retention (complicated) may terminate, like carcinoma, in the complete destruction of the functions of the stomach.

Prognosis.—The prognosis of myasthenia with retention varies according to the duration, the degree, and the complications. If the retention is not great, and general nutrition can be maintained by the establishment of digestive compensation by the intestines, the chances for a complete cure are favorable. In many cases it is only possible to convert the retention into the stagnation form of the disease and to maintain the body in a fair nutritive condition by persistent and carefully regulated digestive hygiene. The prospect is darker where gastritis coexists, particularly the hypersthenic form. The prognosis is worse where the intes-

tines are also diseased, for the insufficiency of the intestines determines, to a large extent, the future. The quantity of urine passed in twenty-four hours and the quantity of food retained in the morning after the test-supper are also prognostic guides. The situation is all the more serious where secondary diseases exist, due to auto-intoxication, or where there is a serious independent trouble. It is safe to give a guarded prognosis in all cases, and watch the results of treatment for encouraging signs.

Treatment.—The objects sought by treatment are: (1) The improvement of the motor function; (2) the prevention of starvation; (3) the control of fermentation; (4) the treatment of the gross symptoms, of auto-intoxication, and of the complications.

To increase the power and the efficiency of the muscular layer, the same remedies which are valuable in the treatment of myasthenia with stagnation may be used, including massage, electricity, hydrotherapy, strychnin, quinin, and ergot. Massage should not be used to evacuate the stomach until the excessive fermentation is under control, and it may then be employed regularly four hours after the evening meal. The proper time to employ electricity is after lavage, the cervicogastric, spinogastric, and von Ziemssen methods, with cathodal excitation, low density, and strong currents, and with short sittings, being preferred. Electric treatment may also be given with advantage one or two hours after breakfast, the action of the remedy on the secretion and on the movements of the stomach being used to promote digestion and the evacuation of the stomach. In addition to the uses of water directed against the myasthenia, stomach washing must be employed against the retention and the fermentation. Another remedy, required by the thin and weak abdominal walls and the overweighted stomach, is a snugly fitting abdominal bandage, to give mechanical support. All constriction about the waist should be removed. The mechanical support and the removal of compression are valuable aids to the evacuation of the stomach and the bowels.

One of the most difficult objects to attain is the nourishment of the patient. To maintain the balance of nutrition all our resources, if necessary, should be brought to bear; including a proper diet, whose utilization is protected and aided, and supplemented, when necessary, by rectal feeding. The remedies which control fermentation and which aid the evacuation of the stomach contribute to this end.

The diet of retention myasthenia should be nutritious, in

small bulk, resistant to fermentation, sufficient to support nutrition, and capable of utilization by the intestines. Digestive compensation is dependent largely on the integrity of the functions of the intestines, the retained gastric contents being unabsorbed and serving only as food for the large quantity of germs.

Sweets should be excluded, and fat (only in the form of fresh butter) should be given in moderation. Oil rubbings may be employed to furnish part of the fat required to support nutrition. Meats, fish, and the whites of eggs form the staple articles of diet, and should be given freed from all indigestible matter and finely divided. The meat jellies, resisting fermentation and filling a deficiency created by the sweets, are very valuable. Rice and the preparations of wheat are the best cereals, and dry toasted bread may be allowed. Dry meat powder and "somatose" are very concentrated foods, and where too little food is eaten they may be introduced into the stomach through the tube at the end of the morning lavage. Fruits of all kinds should be excluded, and cases are hardly known where milk agrees. A glass of water should be allowed with each meal, and a little old whisky or brandy may be permitted if the disease is not so far advanced that gastric absorption is reduced to zero. Alcohol is absorbed by the normal stomach, and promotes also the absorption of other food products.

As a rule, it is best to furnish only three, or at most four, meals a day. The frequent administration of small quantities of food robs the stomach of the possibility of repose, and is a good way to make sure of gradual gastric retention. But in a few cases the stomach is capable of evacuating small quantities of fluid food, and retention may be avoided by giving the nourishment frequently and in small quantities. This power and peculiarity of the stomach is more often met with in the beginning of myasthenic retention, and where the stomach is not sensibly enlarged. All the food should be given in a state of fine division, so as to favor its evacuation by the stomach and its utilization by the intestines, and to facilitate thorough lavage.

Since its recommendation by Kussmaul, stomach washing has become a classical remedy in gastric retention. The technic of the procedure has already been described. In the treatment of myasthenic retention the most favorable time for its employment is in the early morning before any food has been taken. This is usually sufficient to control the fermentation; it protects the intestines, removes but little food

that is likely to prove of nutritive value, and the stomach begins the work of the day fresh and clean. The contents of the stomach should be expressed and the stomach should be thoroughly washed out with warm boiled water, to which an alkali or one of the many antiseptics recommended may be added. The following are the antifermentative solutions most often used: Salicylic acid (1 : 1000), boracic acid (1 : 100), borax (five per cent.), benzoate or salicylate of sodium (one per cent.), permanganate of potash (1 : 1000). After thoroughly washing out the stomach with warm boiled water, a pint of a borosalicylic solution (acid boracic, ℥j; acid salicylic, grs. xx; aq., Oj) should be allowed to flow in, be brought thoroughly in contact with the mucous membrane by lowering and raising the funnel several times, and should then be allowed to flow out. This is a very efficient solution. The stomach should be left empty. In a few cases, in order to secure sleep, to give a long rest to the stomach, and to control obstinate fermentation, it is necessary to perform lavage in the evening—as late as possible after the evening meal. The stomach may be washed out thoroughly (the contents being previously expressed) with a warm alkaline solution, and ℥ss each of salicylate and bicarbonate of soda, dissolved in a few ounces of water, may be left in the stomach during the night. This alkaline antifermentative solution readily destroys yeast and sarcinæ. On the following morning the intragastric douche may be employed, using then, also, the borosalicylic solution. The excessive fermentation will soon be controlled by this method, a daily morning lavage being thereafter sufficient to check the germ growth. Rarely, it may be necessary to combine with the lavage functional repose of the stomach and rectal feeding for a few days.

With some authors the administration of antifermentative drugs is in great favor. Of these drugs the following may be mentioned, and given without much hope of benefiting the patient: Resorcin resublimat. (Merck's), grs. iij to v; bismuth salicylate, grs. v; salol, grs. x; beta-naphthol, grs. iij; sodium salicylate, grs. v; salicin, grs. x; creosote, gtt. iij; sodium benzoate, grs. v; ichthyol, gr. j; aq. chloroformi, ℥ss. These drugs may be prescribed in the above doses, three times a day after meals, in various combinations.

The treatment of myasthenic retention by the systematic use of these remedies often gives relief, and may produce a complete cure. But the result is not always so satisfactory, and, even at the price of their continued daily employment, the patient may be insufficiently nourished and unrelieved of

his sufferings. As a last resource, the aid of a surgeon may be invoked.

The operation of Bircher is yet in its infancy, but where the motor power is not completely destroyed it would seem to be worthy of further experimentation. The object of the operation is the diminution of the size of the stomach, the elevation of the greater curvature by infolding the anterior wall in the line of the long axis of the stomach, and the union of the peritoneal edges of the infolded part along the lesser curvature. From this line of union hangs the anterior wall, the stomach being unopened by the operation. Prof. Weir also employed the same method to reduce the size of a "dilated" stomach in a patient suffering from retention, some time after the performance of gastro-enterostomy, making several folds in the direction of the long axis of the stomach. The preferable operation is gastro-enterostomy.

SECTION V.

THE ANATOMICAL DISEASES OF THE STOMACH.

CHAPTER I.

GASTRITIS.

Inflammation of the internal glandular lining membrane of the stomach may be acute or chronic.

ACUTE GASTRITIS.

Acute gastritis presents three distinct clinical and pathological forms, each possessing a characteristic genesis and evolution, and demanding special treatment. These three forms of acute gastritis are: (1) Simple gastritis; (2) mycotic gastritis; (3) toxic gastritis.

Acute simple gastritis is an afebrile inflammation of the mucous membrane of the stomach excited by neither microorganisms nor poisons. A gastritis excited by germs developing in the cavity of the stomach or in its walls is mycotic. Toxic gastritis is an inflammation of the internal layer of the stomach, excited by the accidental or intentional administration of a poison. Many of these poisons are included among the articles of the materia medica.

I. ACUTE SIMPLE GASTRITIS.

Acute simple gastritis is a very frequent disease; it may be very mild and transient, and may pass away after a few meals have been digested with difficulty and discomfort; or it may be expressed by signs of greater irritation, such as nausea, vomiting, and depression. It is often described as indigestion or as acute gastric catarrh.

Etiology.—Two factors play a more or less important part in its causation: bad alimentation and a diminished resistance

to disturbing influences on the part of the organ. One or both of these morbific causes may be active, the inflammation being the result of excessive irritation or of a morbid sensibility of the mucous membrane to even normal excitation.

The soil may be prepared for the development of the disease in many ways, all of which act through a derangement of one or more of the physiological factors of digestion. The influence of heredity has been frequently asserted without conclusive reasons; common family dietetic faults constitute a stronger predisposition. "I inherit my bad stomach, and my family as far back as I can go has been dyspeptic." There may be some consolation in posing as the victim of uncontrollable circumstances, but a man's stomach, as regards its primary diseases, is what he has made it. We readily admit, however, the inheritance, and also the influence of the catarrhal and the uric acid diatheses. Exposure to excessive heat or cold during the period of digestion, mental or physical fatigue, intense moral anxiety or depression, etc., may render the stomach relatively incapable, and may create the morbid opportunity.

The inflammation may often be attributed to very palpable errors of diet—the food and drinks, on account of their quantity or quality, or their delayed evacuation, producing excessive stimulation. The disease begins frequently after a relatively rich and rapidly eaten meal. The chemical irritants may be proper to the food or drinks, or may be developed in the uneaten food by fermentation, or added by the cook with an excessive desire to please the palate. Or the excitant may be thermal, the food or drinks being too hot or too cold. The most common cause is the irritant quality of the diet—mechanical, chemical, and thermal. Alcohol may often be incriminated, and cold beer seldom fails to produce the disease. Many articles of the materia medica in therapeutic doses are capable of producing the inflammation—arsenic, the iodids, the bromids in strong solution, salines, and excessive quantities of common salt.

Pathological Anatomy.—Acute simple gastritis not being a fatal disease, an opportunity to study its pathological anatomy is very rare. The inflammation is superficial, and the signs are soon effaced by postmortem changes. The circulation phenomena may be very different after death from those present during life, and nothing may be left except ecchymoses to recall the intense, active hyperemia. But observations through a gastric fistula, experiments on animals, and a few

cases examined soon after death due to intercurrent accidents, enable us to describe the morbid anatomy.

The lining membrane is covered with a layer of tough mucus, which may be here and there tinged a rose color. The mucous membrane is red in patches, studded here and there with punctate superficial hemorrhages, and swollen. Beaumont described small vesicles, separated by patches of deep congestion, in the stomach of Alexis St. Martin during an attack of acute gastritis. These appearances are most pronounced in the region of the pylorus, and spread to a variable extent over the internal surface. The surface cells are swollen and distended with mucus, desquamated, and mixed with leukocytes in the mucus covering the surface. The chief cells are granular, stain deeply, are often shriveled, and may be seen lying free in the lumen of the glands, which may be filled with degenerate granular matter and a fluid resembling mucus. The border cells appear unchanged. The capillaries are enlarged and distended, and there is small-cell infiltration, particularly along the venous radicles, and extending even into the submucosa. After the acute inflammation has lasted a few days, the infiltration of the interglandular spaces with small lymphoid or embryonic cells may be greater; and the infiltration may be diffuse, or it may be limited to the superficial or to the deeper parts of the mucous membrane; or it may occur in small, sharply limited nodules, very rich in small cells; or in larger patches which are gradually lost in the adjacent healthy parts. These infiltrating embryonic cells stain lightly, and contain a very large nucleus. Mixed with these small cells is a large or small number of wandering leukocytes, accordingly as the inflammation is more or less intense. The endothelium of the lymphatics may be cloudy, swollen, and undergoing desquamation. It is more than probable that the histological changes may be confined to the surface cells in very mild cases, and the exudation and the desquamation may be accompanied simply by active hyperemia. In some cases the inflammation may be almost exclusively parenchymatous, when the most marked lesion is degeneration of the chief cells. In other cases the inflammation may be interstitial and characterized by infiltration with embryonic cells and leukocytes. Rarely, the inflammation may be diffuse.

Clinical Description.—The disease begins during the period of digestion, without chill, pain, or fever. The patient feels dull, uncomfortable, weak, and indisposed to work. The stomach is heavy and full, the respiration shallow, and the

pulse may be large and compressible or small and slightly increased in frequency. There is slight nausea, often a dull frontal headache; and there may be ringing in the ears, or vertigo, increased by movement or by the upright position. The face is pale, the expression haggard, and the extremities cold. The stomach may empty its contents into the intestines, and these symptoms subside; or the nausea may increase and lead to vomiting, which the patient may instinctively excite. During the period of digestive rest there is physical and mental depression, the appetite is lost, and there may be disgust for the usual food, and the patient may long for sour or spicy articles. The mouth is sticky, and the large white tongue possesses a brownish, thick coating of mucus, bacteria, and epithelium over its base, while the reddened edges bear the imprint of the teeth. The sleep is broken, the bowels constipated, or there may be one or two diarrheal movements containing mucus and producing burning and tenesmus. The urine is small in quantity and deposits urates on standing. The trouble may subside in a few days under the influence of rest and a small quantity of non-irritating food; or the disease, under improper treatment, may become complicated with gastric fermentation and be transformed into mycotic gastritis; or the subjective digestive symptoms may recur with diminishing intensity until recovery is complete.

Throughout the course of the disease there is no gastric pain, but diffuse tenderness, and no fever, except a slight possible increase of temperature over the stomach during the period of digestion. But children may show a rise of about one degree in the general temperature.

The vomit is copious and consists of undigested food, particularly meat, mixed with mucus; it may contain no free HCl, and it may be neutral or alkaline in reaction, or slightly acid on account of the hardly noticeable fermentation. The organic acid is usually butyric. Or the vomit may be excessively acid (H+C), and contain bile and mucus stained with fresh blood. The special secretion of the stomach may be suppressed on account of the congestion and the degeneration of the chief cells. The excessive secretion of mucus and the inflammatory exudation diminish the quantity of free HCl, if any is secreted. Or secretion may be active as an expression of the irritation, the inflammation being then probably superficial or interstitial.

Diagnosis.—The beginning during the period of digestion, the short duration without fever or noteworthy pain, the subsidence of the symptoms after the evacuation of the

stomach or the digestive tube, and the characteristics of the vomit,—leave little doubt as to the nature of the disease.

Treatment.—The treatment of simple acute gastritis is best conducted without the use of drugs.

If the patient is seen before the stomach has been emptied by vomiting, a glass of water at 90° F. may be administered and vomiting excited by the finger in the pharynx. It is seldom necessary or advisable to use an emetic or lavage. The stomach, once empty, is left completely at rest for several hours, a little barley water, lemonade, or egg water, being used to quench thirst.

For twenty-four to thirty-six hours the stomach should be given functional rest. At the end of this time a little thoroughly cooked cereal, or meat juice, or preparation of milk may be ordered; then the more digestible finely-divided meats, a little dry toast or cracker, or crust of roll. At the end of two or three days the patient can be given an ordinary mixed diet composed of the foods which have little physiological action on the stomach. To the cereals and meats, butter, vegetables, and, last of all, sweets, may be added in relation to the rapidity of recovery and the return of the functional power of the stomach, which, after the first day or two, may be improved by small doses of strychnin or of nux vomica.

Recovery may be hastened and the nausea controlled by the cold compress, applied according to the method of Winternitz.

The constipation should be relieved by an enema of warm water, to which a teaspoonful of glycerin may be added. If the irritant has passed into the intestines, a dose of calomel may be given with benefit. The evacuation of the stomach, the compress, the functional rest, the protection of the stomach against irritation, and the relief of the constipation by injections are the remedies most conducive to rapid and complete recovery.

II. MYCOTIC GASTRITIS.

Mycotic gastritis may exist in three forms. In one the characteristic sign is acute gastric fermentation, the result of which is an inflammation of the mucous membrane. Acute gastritis may also be produced by eating food which has undergone putrefaction. The attacks begin like acute poisoning, with headache, vertigo, nausea, vomiting, dyspnea, collapse, delirium, and with other symptoms which vary according

to the dose and the nature of the putrefaction poisons. But these cases are not cases of gastric putrefaction, which never occurs except as a result of gastric retention. Putrefaction poisons may produce, however, no inflammation of the mucous membrane of the stomach, acting in this respect like alkaloid poisons. These cases of acute food poisoning are not produced by acute gastric putrefaction, and do not constitute a distinct clinical form of gastritis, like the inflammation excited by acute gastric fermentation. Gastritis may also be excited by bacteriological products brought to the stomach by the blood. The stomach may eliminate the poison, and the anatomical changes may begin on the surface, as in simple acute gastritis; or the lesions may develop from the capillaries and involve secondarily the tissues fed by them. Furthermore, mycotic gastritis may be caused, not by germs developing in the contents of the stomach nor by bacterial products brought to it by the blood, but by bacteria which invade and inflame its proper tissues. This latter form is infectious. Consequently the two varieties of primary acute mycotic gastritis are: (*a*) The fermentation form; and (*b*) the infectious forms.

In regard to the fermentation form, it may be contended that the fermentation is the distinctive and predominant characteristic, and that the trouble should be properly named "acute gastric fermentation." The force of this contention may be readily admitted; but the fermentation is the expression of the conditions which permit it—a mere sign. In the genesis of the gastritis it forms the connecting link, and may serve to define it. The morbid evolution has passed into the anatomical stage which is accurately described by acute gastritis due to fermentation.

(A) The Fermentation Form of Mycotic Gastritis.

Etiology.—Gastritis due to fermentation is more frequent in infancy, on account of the peculiar and exclusive alimentation, than at any other period of life. The two extremes of life agree in this respect—that both are more liable to the disease than is the middle period. In old age the stomach has been made slow in action and incapable, or in the course of nature has grown to be so.

Thus we have the two conditions of the active fermentation in the stomach which is the exciting cause and the distinctive characteristic of the gastritis—a diet forming a good culture soil richly infested with germs, and stagnation due to acute

motor insufficiency of the stomach. The disease is most frequent in summer, when the germs of fermentation are most plentiful and virulent, digestion torpid, and the stomach often overloaded with drinks.

Pathological Anatomy.—The anatomical changes do not differ materially from those of acute simple gastritis, but may be more intense and diffuse, in keeping with their production by irritants in solution. Consequently the surface cylindrical epithelium degenerates and desquamates to a greater extent than in simple acute gastritis, while the changes in the glands and in the interglandular tissue are variable.

Clinical Description.—This form of acute mycotic gastritis may be febrile or afebrile. Where the disease runs its course without fever it may present two clinical types—a mild and a severe form.

The mild form may begin suddenly during the night or may be preceded for a day or two by slight headache, belching, and regurgitation of a fluid which is sometimes without pronounced taste, is sometimes sour, and sometimes has an odor of acetic or butyric acid. These premonitory symptoms occur during the period of digestion, and are made worse by fermentable food. Then comes the attack, which usually occurs at night or three or four hours after the chief meal. The stomach is distended, contains an excess of gas, is tender on pressure, but in the mild form there is rarely distinct pain. Nausea is common, with frontal headache, which may be severe, and no relief is obtained until the stomach is evacuated. The vomit is like that of simple gastritis plus fermentation, and is more fluid on account of the more active secretion. The symptoms may subside with the vomiting or the intestines may have received a portion of the fermenting chyme to eliminate by a few diarrheal movements. Convalescence may be complete in four or five days under proper treatment, or may be prolonged by a persistence of the conditions which led up to the attack.

In the severe form the local symptoms are more intense and the constitutional symptoms are more pronounced. But the severe form is not simply the mild form magnified. In addition to the fermentation and the gastritis excited by it, there is also auto-intoxication, and the intestines and the liver become secondarily involved.

The severe form may be preceded by the mild form, or may develop as a sequel of acute simple gastritis, or, more frequently, suddenly, during the digestive period, with premonitory symptoms. There is commonly a slight chill, with little

or no fever, intense headache, nausea, dull or severe pain, and epigastric tenderness proportionate to pressure. These symptoms are increased by additional food or drinks. The stomach is distended, the respiration shallow, the pulse small and frequent, and often hard, and the patient is agitated, anxious, and prostrated. The appetite is completely lost; thirst is often intense. The vomit consists of food, of secretion, of exudation, and of the products of fermentation. The attack is not cut short by vomiting, but the gastric symptoms are temporarily relieved. The vomiting may be frequently repeated, and may be accompanied by severe and painful retching and slight capillary hemorrhage. The stomach evacuates part of its contents into the duodenum, and intestinal irritation and fermentation are added, leading to several large fermenting stools, which may smell musty. The liver is enlarged and congested, the spleen is normal in size, and the slight icterus, which is not rare, is probably the result of the duodenitis extending into or obstructing the common duct, or producing spasm of its sphincter. The severe form may last a few days or one or two weeks, in keeping with the extent and the intensity of the excited inflammation; or it becomes not infrequently subacute and chronic.

The disease begins with active fermentation in the stomach, producing irritation and gastritis, and, extending to the intestines, becomes associated with intestinal irritation or with enteritis.

Diagnosis.—The diagnosis of the fermentation form of mycotic gastritis is dependent on the grouping of the subjective and the objective signs as detailed in the clinical history, and on the detection in the gastric contents of the signs of acute inflammation and of active fermentation. In addition to the chemical and the microscopical examination of the vomit, the fermentation tube test should be made.

The differential diagnosis may present some difficulties; and simple acute gastritis, and the gastric crises announcing the commencement, or occurring in the course of other diseases, may be confounded with it. But active gastric fermentation developing proportionately with the evolution of the symptoms is a definitive sign.

Prognosis.—The prognosis of the mild form is good. The severe form may do lasting injury, and leave chronic gastritis or enteritis as a legacy. In very old or weak patients and in advanced diseases of other organs the prostration may be fatal. It is not the disease itself, but the disease aided by its associations, which kills.

Treatment.—The indications as to the plan of treatment are three: (1) Control the fermentation; (2) relieve the gross symptoms; (3) treat the lesion.

To **control the fermentation** it is absolutely necessary that the stomach shall be completely emptied. This may be imperfectly done by vomiting and by copious drafts of warm water—the vomiting being excited by the disease or artificially. But where it can be used, stomach washing is certainly the most efficient method of cleansing the stomach. In the severe form the intestines are also infected, and there may be auto-intoxication. A dose of Epsom salts, or of the effervescing citrate of magnesia, will remove both these complications. Afterward, the large bowel should be washed out. The digestive tube being clean, there may be given to quiet the irritation a large dose of bismuth (½ to 1 dram), with which may be combined an anodyne—the deodorized tincture of opium or the phosphate of codein. The stomach should be kept free from food, which would serve as a germ culture soil, for from twelve to twenty-four hours.

To **relieve the gross symptoms**—the nausea and vomiting, pain and prostration—much will already have been done by cleansing the digestive tube and by the administration of the bismuth and the opiate. But pain may require the earlier hypodermic administration of morphin, and, if the prostration be very great, camphorated oil (1 gr. in 10 minims of oil of sweet almonds) or strychnin should be given hypodermically. After the copious vomiting, dry champagne may be ordered with benefit.

The **treatment of the lesion** consists in the use of the same remedies as in acute simple gastritis—functional rest and favoring of the stomach, rest in bed, the compress, and the administration of strychnin as the reparative process advances.

(B) Infectious Forms.

Acute gastritis may be produced by pathogenic germs which grow on the surface of the mucosa, or which develop in the structures that compose the gastric wall. Growths of favus, of thrush, and of the larvæ of insects on the mucous membrane of the stomach are curiosities of medicine. Trichinosis and actinomycosis may affect the stomach. Acute gastritis, with sometimes pseudomembranous formation, may occur in scarlatina, variola, typhus, pyemia, and septicemia. The bacillus of anthrax, the streptococcus erysipelatosus, and the bacillus of influenza may produce their peculiar tissue

alterations in the gastric mucosa. Diphtheric gastritis is very infrequent, and is always secondary to diphtheria of the upper air-passages. But only two forms of infectious gastritis are of practical interest: purulent gastritis and gastric fever.

(1) PURULENT GASTRITIS.

Gastritis accompanied by diffuse or circumscribed suppuration, due to the invasion of the gastric wall by the pus-forming bacteria, is a rare disease. In going through the literature of the subject, only 65 well-authenticated cases have been found.

Etiology.—The disease is due to the development within the gastric wall of the pus-forming bacteria. The majority of the cases have occurred as secondary infections—in pyemia, puerperal fever, gastric ulcer, gastric carcinoma, purulent meningitis, variola, and scarlatina. In other cases ordinary lesions of the stomach, such as traumatism and gastritis, have afforded the opportunity for bacterial invasion. About five-sixths of the reported cases occurred in men.

Pathological Anatomy.—The pathological anatomy is that of purulent cellulitis, the pus being diffused or collected in the submucosa, particularly about the pylorus. The process extends along the lymphatics and the blood-vessels to the peritoneum and to the mucous membrane. The gastric wall is swollen four or five times thicker than normal, and is infiltrated with inflammatory products. The pus may be diffused, or may be collected in a single abscess, or in multiple abscesses. As a rule, there is only one abscess, which may vary in size, and is usually situated near the pylorus. The involved peritoneum may be covered with a thick, fibrinous false membrane, or adhesions may be formed with adjacent parts, or the peritoneum may be but slightly or not at all inflamed. The mucous membrane may be but little changed, or may be intensely red, hemorrhagic, ulcerated, or perforated by many small openings, extending through the interglandular connective tissue down to the pus-infiltrated submucosa. On pressure, pus oozes through these openings as from a saturated sponge. The mucous membrane may be deprived of its blood supply, and the slough, falling away, may leave large or small ulcers. The interglandular tissue is infiltrated with embryonic cells and wandering leukocytes. There may be, rarely, thrombosis of the gastric, hepatic, and pulmonary veins. The muscular layer may be but little involved, but the connective tissue is infiltrated with pus, the bundles of

fibers are separated, and the muscular fiber is degenerated or disintegrated. The abscess may open into the stomach, or the peritoneal cavity, or the pleural cavity; or may burrow through adhesions into adjacent organs.

Clinical Description.—The disease usually begins suddenly, with a severe chill, though in some of the cases there have been, for two or three days preceding the commencement, symptoms of digestive disorder or symptoms of the primary disease of which the purulent gastritis is a secondary infection. The chill is accompanied by high fever, which is usually remittent, ranging from 102° to 105° F.; and the rigor may be several times repeated during the evolution of the disease. There are great prostration and extremely severe epigastric pains, which may not be increased by pressure; and the stomach may be retracted in the commencement, but soon relaxes, and is distended with gas. There is nausea, repeated vomiting, pinched countenance, prostration, and the symptom-group may suggest acute poisoning. Sooner or later peritonitis develops and adds its local and general signs to those given by the intense cellulitis. The peritonitis may remain localized or may become general, and in the pyemic cases the pericardium and the pleura may also be affected. The brutal beginning is sometimes followed by a short period of calm, after which the symptoms become continuous or remittent, and usually subside again before the fatal termination in collapse or in coma. Vomiting may not appear until the second day; it becomes frequent, and finally diminishing in frequency, subsides during the calm preceding death. The vomit consists of undigested food, mucus, inflammatory products, blood, and bile, but little or no pus, except toward the end, when the abscess may have opened into the stomach. The bowels may be obstinately constipated, but diarrhea is more common. Where circumscribed, the abscess may present a palpable tumor, which is usually tender on pressure. In the circumscribed form the pain and fever may subside for a few days when the tumor appears, to be followed a few days later by hectic fever and the signs of peritonitis or of perforation into the stomach or into some other cavity.

The general symptoms are even more intense than the local signs—extreme prostration, anxious and pinched countenance, frequent, small, irregular pulse, delirium with suicidal impulses. Tetany may also develop.

The duration of the disease varies from three or four days to as many weeks. Where the suppuration is circumscribed, the local and the general symptoms are less intense, the fever

becomes ultimately hectic, and the duration is much longer than in the violent, rapid, and diffuse form. The average duration is about one week.

The disease ends fatally in 95 per cent. of the cases. That complete recovery is possible has been demonstrated by anatomical preparations (Dittrich, Deininger).

Diagnosis.—The disease has no characteristic sign; the evolution and the combination of symptoms are not distinctive. The diagnoses of intestinal obstruction, of peritonitis, of abscess formation in other parts of the abdomen, have been made. Epigastric resistance to palpation or a tumor which becomes less and disappears after the vomiting of pus may be supposed to be characteristic; but this combination of signs is rare, and might be due to an abscess which had burrowed and opened into the stomach. Corrosive poisoning is easily excluded by the absence of the distinctive effects of such poisons on the mouth and throat.

The development of the symptoms in the course of one of the infectious diseases which it sometimes complicates may create a suspicion. It may be confounded with acute pancreatitis or pancreatic abscess, with perigastritis developing in the course of perforating ulcer of the stomach, with hepatic abscess, and with purulent cholelithiasis.

Treatment.—The treatment is purely symptomatic so far as the medical management is concerned. Where the disease is suspected and attributable to pus formation and to local peritonitis, an exploratory laparotomy is indicated. Surgical treatment might be successful where the pus is collected in abscesses, and a tumor, developing with the symptoms of acute purulent inflammation, should be explored with a needle, with a view to operative intervention.

The disease usually goes undiagnosed before death, and the treatment has been that of the disease for which it was mistaken. Functional rest, opium to relieve pain, control of the excessive fever, stimulants—summarize the symptomatic treatment. Some recommend quinin.

(2) GASTRIC FEVER.

The existence of this form of infectious gastritis is denied by some authors, who do not admit that the disease is specific, but contend that the cases reported as such are either food intoxications, severe forms of common gastritis, or abortive attacks of well-known infectious diseases, such as typhoid fever and cholera.

While the specific nature of the disease awaits the discovery of its pathogenic germ, clinically the gastritis is neither simple, nor fermentative, nor toxic; but in its pathology and evolution is distinct, and analogous to the bacterial infections. It occurs in persons who have already been affected with typhoid fever, from which it is clinically clearly distinct. The fever is continuous, the spleen is not palpable, and the course is uninfluenced by quinin, the administration of which increases the gastric symptoms—which is not the case where the plasmodium malariæ exists in the blood. It bears a close resemblance to the gastric form of influenza, but in our experience usually occurs isolated in families and is not contagious. The disease is more frequent in spring and in autumn, and occurs more often in the middle period of life than in infancy, childhood, and old age. It occurs isolated, and endemics have been reported.

Clinical Description.—The beginning is like that of most infectious diseases of mild type—general discomfort, a slight chill, fever, headache, and pains in the extremities, in the back, and in the epigastrium. The general precede the gastric symptoms, and develop independently and out of proportion to the gastritis. Several hours after the commencement there are pains in the stomach, epigastric tenderness, nausea, and vomiting. The vomit consists of an alkaline fluid containing mucus, and, incidentally, sometimes bile and undigested food. The bile is usually absent, except after retching, and the development of the gastric symptoms is in no relation with the diet or with the period of digestion, and there is no gastric fermentation.

The fever attains its highest point within forty-eight hours, and oscillates between 100° and 103° F., with a morning remission of about 1° F.; it continues, uncontrolled by quinin, for ten or twelve days, and drops, with a greater morning remission, in two or three days to the normal point.

In addition to the initial chill, slight rigors may occur in the course of the disease. The bowels may be constipated, but there are usually a few diarrheal movements, which have none of the characteristics of the typhoid stool. The pulse is rapid, the patient restless, and delirium may occur in the course of the disease. Insomnia is the rule. The nervous symptoms, apart from the often severe initial pains, continue more or less throughout the disease—headache, restlessness, insomnia, vertigo, tinnitus aurium, prostration, and, with the high temperature, sometimes delirium.

Diagnosis.—This form of infectious gastritis may be con-

founded with typhoid fever. But the step-like rise of the temperature during the first week, and its slow remittent decline during the third or fourth week, the enlarged spleen, the characteristic stool, the ileocecal stagnation and gurgling, the Widal serum sign, and the eruption are all absent. A few bronchial râles may exist, as in typhoid fever; but the gastritis is an essential and distinctive sign, and the spleen is never palpable.

From the fermentation form of gastritis and the severe form of simple acute gastritis it differs in the absence of fermentation and of a relation with alimentation and digestion, and in the predominance of the general symptoms. In gastric fever the disturbance of the system is much greater than the disease of the stomach would naturally produce, and the general and the local symptoms bear no close relation to each other. The evolution of the disease, which is in itself distinctively characteristic, is not controlled by treatment.

Treatment.—The treatment is that of an infectious disease the course of which can not be abridged by drugs. Tepid sponging, the compress, and a fluid diet are indicated to control the fever and the gastric symptoms. Calomel may be given in the beginning, and often exercises a good influence on the symptoms and the temperature. The nervous symptoms may require phenacetin, but it is best not to disturb the stomach with drugs oftener than can be avoided.

III. ACUTE TOXIC GASTRITIS.

Acute toxic gastritis is an inflammation of the mucous membrane of the stomach excited by the action of certain poisons introduced into the stomach from without.

The poisons which produce an injurious effect on the stomach may be formed within the body or may be introduced from without. The endogenous poisons are brought to the stomach by the blood. We have nothing to do here with the diseases of the stomach (among which may be gastritis) due to the retention or the excessive formation of normal nutritive or secretory products, or due to the abnormal substances formed by disease or by bacteria within the body.

Acute toxic gastritis may be produced by alcohol, by tobacco, and by many of the drugs extensively used in the treatment of disease. The essential oils and resins, the iodids, the preparations of mercury, the bromids, the salicylates, arsenic, purgatives, iron and its compounds, and a large

number of other drugs, when given improperly, or for a long time, produce gastritis. But it would not be wise to say more of the serious injury which is often done while conscientiously endeavoring to do good. Acute toxic gastritis is, however, often produced by poisons which have been taken by accident or for suicidal purposes, or which have been administered with forethought and malice. Some of the most common forms of it will be briefly described.

All poisons are not capable of exciting an acute gastritis. As regards their action on the stomach, poisons may be divided into three classes.

1. Those which enter into chemical combination with the tissues, either dissolving the cells, as do the caustic alkalies, or coagulating the protoplasm, as does alcohol, or uniting with their constituents, as do phosphorus and the mineral acids. These are chemically destructive poisons.

2. Those which irritate and excite inflammation of the glandular layer.

3. Those which produce no anatomical change in the mucous membrane perceptible during life or visible after death. This class consequently plays no part in the etiology of toxic gastritis.

(*a*) **Poisonous Acids.**—The stronger acids brought in contact with the mucous membrane of the stomach directly, produce death of the tissues by dehydrating, coagulating, or combining with the constituents of the cells, for which they have strong chemical affinities; and by irritation of the adjacent parts they excite inflammation, which may be so intense as to lead to local death. The separation of the necrosed tissues is a reactive process, which may be followed by cicatricial healing, or by regeneration, if the action has been superficial.

Pathological Anatomy.—The stomach is the center of the destructive changes produced by swallowing corrosive acids, being the place where they are first arrested, and where they long remain. The action on the stomach is conditioned by the quality, the concentration, and the chemical affinities of the acid, and by the quantity and the nature of the contents of the stomach at the time when the poison is swallowed. In small quantities the action is most intense and may be confined to the region about the cardia and the lesser curvature, along which the acid flows to and around the pylorus. Consequently a very small quantity, after recovery by cicatrization, is likely to leave serious and obstructive deformity of the orifices. In larger quantity, the action is more diffused

and general, but is deepest along lines marked by the tops of the folds into which the mucous membrane is thrown by the strong contortions of the stomach. The depth of the direct destruction varies according to the quantity, the nature, and the duration of the action of the poisons. The coagulation necrosis may extend rapidly through the entire thickness of the wall, may undergo dissolution, and thus produce perforation. The coagulation of the blood may extend far into the veins, reaching the liver, the right heart, and the lungs. Such direct and extensive destruction and coagulation are rapidly fatal. Where the violence is less brutal, the direct chemical necrosis is more superficial, and the reactive inflammation may separate the slough, and, with granulation formation, accompanied by free suppuration and by bacterial infection, may ultimately end with a cicatrix which may inclose the remains of glands. Or the inflammation, intense and hemorrhagic, may end in local death and an extension of the loss of substance. The necrosis may be superficial, but may occur in patches; and recovery may leave a deformed stomach, with preservation of more or less of its glandular structure.

Clinical Description.—The symptoms are due to the local action of the poison on the tissues with which it comes in contact, to the diminished alkalinity of the blood, and to the toxic nephritis.

The local symptoms develop immediately, and are extremely violent. The pain is burning and horrible in the mouth, throat, the beginning of the esophagus, at the point where it passes the left bronchus, immediately above the cardia, and in the stomach. The stomach is in tonic spasm, and the excruciating pain radiates over the abdomen. The patient writhes in agony, the pulse is small and weak, and the body covered with a cold perspiration. A brownish saliva flows from the mouth, and intense dyspnea may be present if some of the acid has entered the larynx or if there is a rapidly developed edema of the glottis. Saliva, which may be colored with altered blood, flows from the mouth; and vomiting, aided by unquenchable thirst, dysphagia, intestinal pain, mucous and bloody diarrhea, and tenesmus, adds to the miseries of the patient, who sinks rapidly into collapse, and dies within from two to twenty-four hours.

The local symptoms may be less violent, and the patient may live long enough for the blood and renal symptoms to develop. If the stomach is not already empty, the vomiting may be delayed. The vomit consists of the contents and mucus, colored with hematin, and of the acid swallowed.

The urine, which is passed with pain, contains serum albumin and globulin, hematin, and casts.

The diminished alkalinity of the blood may add to the nervous symptoms. If a large dose of an acid be given to a herbivorous animal, it falls at once into a stupor, and dies before the blood becomes acid. Carnivorous animals and man have a greater power of neutralization; but the diminished alkalinity of the blood will produce somnolence, stupor, or coma, in proportion to its degree.

The symptoms due to the renal lesion are those of acute parenchymatous nephritis with intense irritation, but no edema develops.

The subsequent course is marked by the combined local, nervous, and renal symptoms. The temperature, subnormal in the beginning, rises a few degrees. The dangers do not end with recovery, for the deformities which result from cicatrization may compromise existence.

Diagnosis.—The sudden beginning, and the violence of the symptoms in the midst of health, would suggest poisoning. But the patient may already have been ill before the acid was swallowed. The excessive acidity of the vomit would create suspicion. The diagnosis of corrosive poisoning will be given by inspection of the mouth, and the color of the necrosed spots may suggest the particular acid. The symptom-group, and the examination of the urine and the vomit, reveal the particular acid to which the gastritis and its treatment stand in direct relation.

In sulphuric-acid poisoning the local action is very destructive, and grayish-brown eschars are found, which extend deep into the tissues, the surface being hard and parchment-like; the toxic nephritis is likewise intense, and the duodenum and the small intestine may be inflamed and ulcerated in spots. The cicatrices are large, and the prognosis must be guarded. Even after resolution the strictures offer little hope of relief by surgical operations. The fatal dose, on an empty stomach, is about five grams. Perforation is frequent.

Nitric acid produces lesions similar to those produced by sulphuric acid, but perforation is less frequent, and the eschars are brown on account of the formation of xanthoproteinic acid. The fatal dose is six to eight grams.

Hydrochloric acid produces a grayish eschar on the mucous membrane and no cauterization in the corners of the mouth, or but rarely. The eschar may be yellowish, like that of nitric acid. The toxic nephritis is rare, and strictures are not so frequent with hydrochloric and nitric as with sul-

phuric acid. But the nervous symptoms are more prominent, and death may occur in collapse, in convulsions, or in coma before vomiting sets in. Perforation is very rare. Lavage is permissible.

Chromic acid and its salts are very destructive caustics, and the toxic dose is very small—less than ½ of a grain. The color of the eschar on the skin and the mucous membrane is yellowish-white, becoming grayish-brown, and the vomit, which is rapidly produced, is colored yellowish-red or, by oxidation, may be green. It produces severe abdominal pain, diarrhea, cramps, collapse, cyanosis, and death; or the patient may recover after showing yellow sclerotics, toxic nephritis, and anuria. The temperature may rise and the uremia may be the cause of death. The nephritis often becomes chronic. The alkalies neutralize the chromic acid, but form poisonous salts. Acetate of iron is the best antidote. The stomach may be washed out with a $\frac{1}{10}$ per cent. solution of nitrate of silver, as the silver chromate is insoluble. The intestines are more involved than in other acid poisonings, and with the gastritis is associated enteritis, often with large ulcers, particularly in the colon.

Acetic acid may also produce gastritis, and is of special interest on account of its wide-spread use as a condiment and its production in the stomach by fermentation. Concentrated, it is as destructive as the strong mineral acids, and produces a white eschar, cicatricial deformities, and, in large doses, nephritis. It is oxidized and eliminated as carbonates, rendering the urine neutral or alkaline. Like tartaric and lactic acids, long continued in small doses, it produces chronic gastritis.

Oxalic acid produces a whitish eschar, surrounded by a zone of intense inflammation, which separates with difficulty. The vomit is seldom colored, and there is no hemorrhage. This acid has been found in the stomach as a bacterial product. It produces, in addition to the local symptoms, violent convulsions and rapid death, through its action on the nervous system. It excites also toxic nephritis, anuria, colitis, and enteritis, with punctate hemorrhages and ulceration.

Formic acid excites, locally, an intense inflammation, and in sufficient concentration is also a caustic. It is an oxidation product of gastric fermentation, and consequently is of double interest to the practitioner. It excites nephritis, and is a selective poison for the kidneys unless oxidized into carbonic acid before elimination.

Treatment.—The immediate treatment consists in the administration of the chemical antidote. This is not the same for all acids. Magnesia usta is the best antidote for the mineral acids, but other alkalies may also be used. The carbonates of the alkalies should be avoided, on account of the danger, in severe cases, of rupture of the necrosed stomach. But delay is dangerous, and may be fatal, and whatever is at hand should be given—as soup, milk, white of egg, chalk, scraping from plaster walls, or soap.

The aftertreatment consists in the employment of remedies to cure the local inflammations and to control the symptoms. The nourishment and water may have to be given exclusively per rectum. Food given by the mouth should be fluid and non-stimulating, as milk or cereal gruels. Opium may have to be freely used, and healing of the ulcers may be favored by large doses of bismuth. The collapse may be treated with camphorated oil and strychnin, and the coma by increasing the alkalinity of the blood by free hypodermic or intravenous injections of sodium carbonate.

(*b*) **Caustic Alkalies.**—The caustic alkalies, like the caustic acids, produce local death and reactive inflammation. The acids coagulate the protoplasm, and form dry, hard, parchment-like eschars, sharply limited beneath; the alkalies liquefy the protoplasm, and extend very deep into the tissues, and the eschar is softer and like false membrane. The muscular tissue is rapidly disintegrated; the connective tissue is but little affected; and the nerves, except the end plates, are left chemically intact, to undergo degeneration. They do not act, like the acids, on the kidneys and the blood; the vomit is alkaline; the urine, neutral or slightly alkaline; and the reactive inflammation is more diffuse. Cicatricial deformity is a legacy of both forms of poisoning. The treatment consists in the neutralization of the alkali by vinegar or by tartaric acid, and in the employment of medication to relieve the local inflammation and the complications and to control the symptoms.

The symptoms are intense pain, vomiting, collapse, death in convulsions, or from a terminal perforation peritonitis; or, if the acute stage is safely passed, the symptoms are those of the intense reactive inflammation. The vomit is brownish from altered blood, intensely alkaline, tough, and ropy. Ammonia is, in contradistinction to caustic potash and soda, a respiratory stimulant, and may produce a toxic nephritis.

The inspection of the mouth, the symptom-group, and the properties of the vomit give the diagnosis.

(*c*) **Metallic Poisons.**—Arsenic, phosphorus, and antimony do not belong to the caustic poisons. The gastritis excited by them is not in itself characteristic, but all of them lead to fatty degeneration of the parenchyma of the various organs of the body, including the glands of the stomach. Arsenic may produce an intense hemorrhagic gastritis, with the formation of ulcers; phosphorus may excite little inflammation; antimony may cause more intense irritation than arsenic, the epithelium being destroyed or raised here and there in numerous vesicles and pustules.

The gastric symptoms of phosphorus-poisoning are usually delayed several hours, unless the stomach contains fat in some form, and may be very slight. After six or eight hours there may be nausea and vomiting; the vomit is seldom bloody, and contains phosphorus. During the first week the liver enlarges, the spleen remaining normal in size. With the fatty degeneration of the liver icterus also appears, and sometimes fever. During this stage death may occur suddenly from cholemia. A sequel, should recovery take place, is more or less complete gastric atrophy.

Arsenical poisoning is accompanied by more pronounced symptoms, beginning, after an interval of an hour or two, with burning in the mouth, vomiting, and cholera-like stools, cramps in the extremities, and collapse. The vomit is not colored with blood, but contains the poison. If the patient survive the initial stage, nervous symptoms may appear—paralysis of the sympathetic or neuritis. Anuria may result from a toxic nephritis. Recovery is likely to leave permanent effects, from the action of the poison on the abdominal sympathetic and from gastro-intestinal atrophy.

Antimony leaves its trace in the inflammation and vesicles of the mouth, but its gastro-intestinal symptoms are very much like those of arsenic. Influence upon the nervous system, like that exercised by arsenic, is not found in antimonial poisoning.

The diagnosis is dependent on the symptom-group, and on the detection of the poison in the vomit, the stools, or the urine.

Treatment.—The indication in the treatment of phosphorus-poisoning is to remove the phosphorus before its absorption takes place. The only means of doing this thoroughly is with the stomach-tube. The stomach should be thoroughly washed out with warm water; then with a one per cent. solution of sulphate of copper, followed by the introduction of magnesia usta. The stomach is further washed out with water, and a

full dose of Epsom salts is left therein. After the action of the saline, the colon should be thoroughly washed out. In this manner the poison is removed from the digestive tube. Fats must be carefully excluded from the diet. The after-treatment is that of the symptoms and the complications.

Arsenical poisoning is treated also by immediate lavage, to which calcined magnesia is added. The usual antidote (the freshly magnesia-precipitated subsulphate of iron) is then introduced and left in the stomach. The same precaution should be taken to evacuate the whole bowel with a clyster to which glycerin may be added, and followed by lavage of the colon. The aftertreatment is that of the gastro-enteritis, the gross symptoms, and the complications.

The treatment of antimonial poisoning is conducted on the same principles. The lavage water should contain about one per cent. of tannin. The evacuation of the intestines should also receive attention, and the gastro-enteritis should be treated in the usual manner.

CHRONIC GASTRITIS.

The anatomical and clinical conceptions of chronic inflammation of the mucous membrane of the stomach are widely different. If all the cases are to be classified as chronic gastritis in which marked anatomical changes (which assume the characteristics of chronic inflammation) are found at the autopsy, then this is unquestionably the most frequent of all the diseases of the stomach. Chronic gastritis is the almost constant accompaniment of the last stages of all chronic diseases. This terminal gastritis should be placed among the vicious circles of the stomach, along with all cases secondary to a disease of another organ than the stomach, and presenting anatomical or clinical peculiarities.

Gastritis is often a complication of a chronic disease of the stomach; and neither ulcer, nor carcinoma, nor pronounced myasthenia, nor obstructive retention, completes its evolution without the development of gastritis. The clinical conception excludes this complicating gastritis. But ulcer, carcinoma, obstructive retention, and motor insufficiency may appear in the course of chronic gastritis, and only a knowledge of the development of the particular case could enable us to decide which is the primary and which is the complicating disease. Many of the diseases of the stomach are links in the evolution of one process.

Chronic gastritis, as a distinct clinical disease, is a chronic inflammation of the glandular coat of the stomach, not developing in the course or as a consequence of any other chronic disease of the stomach. Before the clinical disease can be established, the characteristic symptoms, the evolution, and the objective signs must be demonstrated, and a chronic associated and causal disease must be excluded. Some authors also exclude from the clinical conception of this disease all cases accompanied by a persistent, excessive, specific secretion. A well-marked clinical form of chronic gastritis (hypersthenic form) is classified and described among the "neuroses" in nearly all the text-books. This is an error.

Etiology.—Chronic gastritis is most frequent in the middle period of life. It is no less common in old age, but is then more often latent.

If it be remembered how many morbific influences have a special affinity for this organ, it will be no wonder that pathologists so rarely find a healthy stomach after the beginning of adult life. Chronic gastritis frequently follows the acute disease. All the infectious fevers and inflammations affect the stomach, and often produce anatomical changes in the mucous membrane. There exists no serious disease of an important organ of the body, or of nutrition, that may not in itself be the efficient or predisposing cause of gastritis. It may become a complication of another anatomical disease of the stomach, or may develop as a consequence of the chronic dynamic affections. But if all these causes were inactive, chronic gastritis would not be a rare disease, on account of the irritants which are introduced into the stomach or which develop in it. All the causes of acute gastritis, acting with moderate violence during a long period,—the products of fermentation, coarse food, excess of condiments, abuse of alcoholic drinks, prolonged use of the bitter aromatic drinks, swallowing saliva impregnated with nicotin or tobacco juice, abuse of purgatives and irritating drugs,—are so many directly acting causes of the chronic disease.

Pathological Anatomy.—The pathological anatomy of chronic gastritis is extremely complex and variable. There is no constant relation between the cause and the morbid anatomy. During the initial period, it is true, the inflammatory lesions due to the irritation of the contents are more pronounced in the superficial part of the mucous membrane, whereas those excited by blood or by circulation changes are most marked in the interglandular tissue and in the submucosa. But the evolution of the pathological anatomy soon

conceals any relation that may have at one time existed with the natural mode of action of the exciting cause. The following diagram shows clearly and at a glance this conception of the evolution of chronic gastritis.

Every case of chronic gastritis displays the common signs of this form of inflammation—disordered circulation, small round-cell infiltration, cell proliferation, and cell-degeneration. Hemorrhagic erosion and superficial ulceration may be associated with any of the forms. The ulcers may be few or innu-

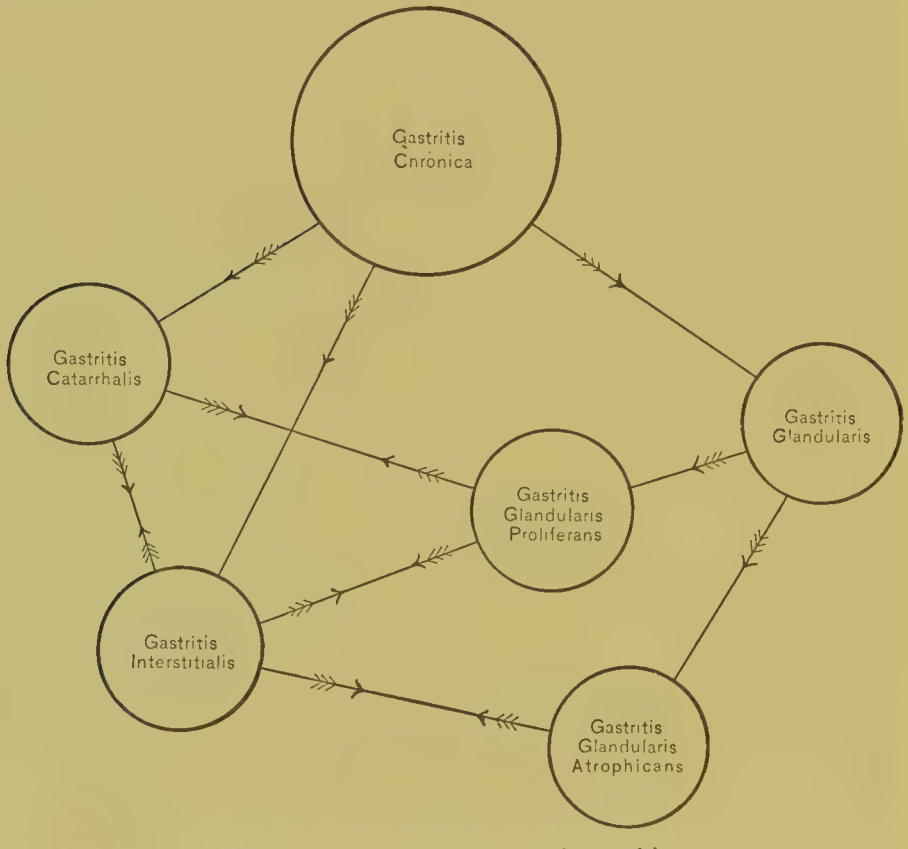


Fig. 19.—The evolution of chronic gastritis.

merable, and vary in size from a pin-head to a small hand. The grayish-brown, thickened mucous membrane may be marked by fine ramifications of dilated blood-vessels. But the inflammatory changes may predominate in particular components of the mucous membrane, and thus produce corresponding pathological forms. Three anatomical varieties of the disease might be described—the catarrhal, the glandular, and the interstitial. Any one of these varieties may terminate in atrophy of the gastric glands.

But this anatomical classification does not represent the clinical forms of the disease, for interstitial gastritis has no characteristic clinical expression and no characteristic functional signs. The diagram which illustrates the evolution of gastritis makes clear the fact that interstitial gastritis may produce catarrhal gastritis, or it may produce proliferating glandular gastritis, or, finally, it may terminate in anadenia gastrica as a result of granular or mucoid degeneration of the chief cells of the gastric glands. Moreover, interstitial gastritis nearly always accompanies both chronic gastric catarrh and glandular gastritis. Interstitial gastritis will, consequently, not be described as a distinct clinical form.

It requires no very great ingenuity to multiply the anatomical forms of chronic gastritis, for the primary forms do not preserve their identity throughout their development. The characteristics of one stage lose their predominance at a further stage of the evolution of the disease. Nature mixes the anatomical forms at various stages, and produces numerous varieties. Furthermore, the gastritis does not develop with equal rapidity over the whole surface of the mucous membrane, nor is it of the same character everywhere. It becomes necessary, therefore, to introduce some general characteristic which displays the predominant condition of the whole mucous membrane. The predominant condition of the mucous membrane is displayed by the functional signs and by the clinical expression.

In a large group of cases the special secretion of the stomach is constantly diminished. The contents of the test-breakfast contain less hydrochloric acid (H + C) and less ferments than normal. Mucus is secreted in excess. There is no spasm of the pylorus, and the stomach empties itself in a normal manner. The appetite is diminished or lost. Pain is rare. There may be nausea, and after meals the stomach feels overloaded. The signs and symptoms are those of depression. The manifestations are not violent. There is no show of strength, but everything is dull and weak. The special acid- and ferment-secreting cells are inactive and degenerate, and there may be active connective-tissue production. Only the cylindrical cells are irritable and secrete a large excess of mucus. The gastritis is catarrhal in its beginning and evolution, and the dynamic expression is asthenic.

Another group of cases are violent in their manifestations. All the signs are those of great irritability. General gastric sensation is acute, and the appetite, when not dulled by pain,

is sharp. Secretion is excessive—excess of acids and ferments and a more than normal quantity of mucus. The pylorus may be closed by spasm, and there may be gastric cramps. The inflammation is glandular and productive in its predominance and evolution. The dynamic expression is hypersthenic.

A third group of cases is characterized by a special causation and by a peculiar evolution. The clinical expression is, unlike that of the two other groups, sometimes latent, sometimes violent. The mucous membrane is exceedingly vulnerable, and gastric secretion progressively diminishes until it almost disappears. The process is atrophic, and the glands and the cylindrical surface cells disappear, and leave a layer composed of connective tissues infiltrated with embryonic cells and leukcoytes.

We recognize, therefore, three distinct clinical and anatomical forms of chronic gastritis—viz : (1) Gastritis catarrhalis chronica, or chronic asthenic gastritis; (2) gastritis glandularis proliferans, or chronic hypersthenic gastritis; (3) gastritis glandularis atrophicans, or progressive atrophy of the gastric glands.

I. GASTRITIS CATARRHALIS CHRONICA, OR CHRONIC ASTHENIC GASTRITIS.

Pathological Anatomy.—The mucous membrane, in chronic asthenic gastritis, is covered with a thick layer of tough mucus mixed with a large number of cells, which are in part desquamated cylindrical cells, in part wandering leukocytes, and in part blood-corpuscles. The color may be reddish-brown, or gray, or darkly pigmented. The coloration of the pyloric region is usually diffuse, while that of the rest of the mucous membrane is mottled. The coloration is due to more or less altered blood pigment and cells mixed with mucus. The mucous membrane is swollen, and the swelling is the result of edema, of infiltration of the connective tissue, and of enlarged, obstructed glands. The round-cell infiltration of the connective tissue may be limited to the superficial or mucous division of the mucosa; or to the interglandular and the subglandular connective tissue; or it may extend to the submucosa and to the muscular layer. The infiltration and swelling is most intense in the pyloric region, and may diminish the size of the pyloric opening. In old cases the mucous membrane may become mammellated, and the prom-

inences may resemble polypi. The surface may be studded with little vesicles on the villosities, or there may be hemorrhagic erosions, catarrhal ulcers, or very small ulcers with the opening of a gland in the center.

The microscopic appearance of a transverse section of the mucous membrane is very interesting. On account of the infiltration of the interglandular connective tissue only about one-half the number of ducts appear in the field, as can be seen in a cut of the normal mucous membrane made parallel to the surface through the mucous layer of the mucosa. The



Fig. 20.—Gastritis chronica catarrhalis. × 240. (Authors' specimen.)

connective tissue is infiltrated with small, round, nucleated embryonic cells, and, although the intensity of the infiltration varies, and may be diffuse or in patches, it always becomes less and less as the eye goes from the surface to the submucosa. Mixed with the embryonic cells are wandering leukocytes and hyaline bodies. The hyaline bodies are the granules of disintegrated leukocytes. Many of the leukocytes possess oxyphile granules, and they wander between the cylindrical cells to the surface, or into the mucous division of the ducts of the glands. Some of them worm

their way directly through the cylindrical cells, and may be recognized therein by their form, by their structure, and by their affinities for stains. The cylindrical cells, which cover the villosities and line the ducts of the glands down to the necks of the tubules, desquamate, reproduce rapidly, become striated along their free ends, and degenerate into beaker cells. The border cells are very few, which is in keeping with the functional inactivity of the glands. The nuclei and the cytoplasm of the young cells stain deeply, and are readily distinguishable from the older and paler cells. The



Fig. 21.—Gastritis chronica catarrhalis et interstitialis. × 115. (Authors' specimen.)

nuclei of some of the cylindrical cells may be seen in the process of nuclear division, and the division may be asymmetrical or typical. Three or four such cells may be seen in the duct of a gland and here and there along the surface of the mucous membrane. Beaker cells are not found in health, but are common in gastric catarrh. The gastric glands may be elongated and dilated as a result of obstruction of their ducts by swollen and desquamated cylindrical cells. The chief cells may undergo mucoid degeneration and vacuolation. The degenerated chief cells may be replaced by cylin-

drical cells, the regeneration proceeding from the cells lining the necks of the glands, which are thus changed from peptic into mucous glands. Catarrhal gastritis, in its evolution, becomes a mixed gastritis, and may terminate in anadenia gastrica. The excessive secretion of mucus and the diminished secretion of acid and ferments are the natural consequences of the anatomical lesions—viz., the irritable hyperplasia and excessive activity of the cylindrical cells, the degeneration and reversion, respectively, of the cylindrical cells into beaker cells and into striated (ciliated) columnar cells, the infiltration of the mucous division of the mucosa, and, to a lesser degree, of the interglandular and subglandular connective tissue. The chief cells may remain normal or degenerate, and the border cells are very few in number, in keeping with the inactivity of glandular secretion. The peptic glands may be converted into mucous glands, or they may disappear; the glandular division of the mucosa being transformed into an infiltrated layer containing, here and there, the remains of a peptic gland, or invaded by the growth and elongation of the portion of the duct which is normally lined with cylindrical cells. Eventually the mucous division of the mucosa may undergo atrophy.

Clinical Description.—The symptoms of chronic asthenic gastritis are very variable. It may be latent for months, or even throughout its anatomical evolution. In its simple form it is a painless disease, and the gastric discomfort, fullness, or weight are the only abnormal sensations of which the patient complains. The other gastric symptoms are belching and sometimes nausea and vomiting. The belching is not a result of gas formation in the stomach, but it is induced, reflexly or voluntarily, to relieve the sensation of fullness. The appetite may be normal, but is more often diminished or easily satisfied. Sometimes sour, spicy, and highly seasoned dishes are preferred, and in a small number of the cases there is disgust for meat. The other symptoms commonly ascribed to chronic asthenic gastritis are due to complications.

On awakening in the morning there may be nausea, and a small quantity of mucus and bile and saliva may be vomited. After the meals the stomach feels full and heavy, and there may be nausea and, very seldom, vomiting. The vomit is thick, ropy, contains no free, but possibly a little combined, HCl, and unchanged food. The digestive discomfort is, with only a few exceptions, worse after meals of solid food, liquids being well borne. There is often repeated belching of swallowed air, bringing with it sometimes into

the mouth a little non-acid fluid. Throughout the day, and particularly during the period of gastric digestion, the patient feels vaguely uncomfortable, and indisposed to mental or physical work.

The failure of digestive compensation occurs early among the poor, on account of the coarseness and the bad quality of their food. The stomach, with its diminished acidity, or even neutral or alkaline contents, is no longer a barrier against the invasion of the intestines by germs. Those who obtain fresh and nutritious food, and protect the intestines by cleanliness, —pure food, pure drinks, sweet mouth and throat,—may live a long period with nutrition perfectly preserved. Whenever malnutrition exists in chronic asthenic gastritis, the cause should be sought for in the alimentation or in the intestines.

Many of the symptoms attributed to asthenic gastritis are due to complicating intestinal trouble. Many of the so-called gastric reflex disorders of the heart and of the capillary circulation have their origin in the intestines. The flatulency, often so great as to force the patient to unloosen the clothing, is in the intestines and not in the stomach, which is pushed up and compressed by the distended transverse colon. The belching of the swallowed air relieves the increased abdominal tension, but the stomach is perfectly free from fermentation and its products.

The course of chronic asthenic gastritis is very slow; at first intermittent, with periods of good health and comfort; it then becomes more continuous, and more rapidly progressive as intestinal compensation fails. Malnutrition now begins, the muscular layer becomes involved in the inflammation or undergoes fatty degeneration, the mucosa has been converted into an infiltrated layer without peptic glands, and the atrophied, flabby stomach may be the seat of severe lancinating pains. Often, however, these pains, which are most common during the period of gastric repose, are located in the colon, the stomach being sweet and empty.

Symptomatology.—The physical signs of chronic asthenic or catarrhal gastritis possess chiefly a negative value—no tumor, no abnormal splashing, normal in size and position, but usually sensitive to pressure.

The functional signs are very important in the diagnosis, the prognosis, and the treatment of the disease. The secretion is characterized first by diminution and then by disappearance of the free and the combined HCl, by diminution and disappearance of the ferments, and by persistent excess of mucus.

The terminal atrophy is never so complete as to give no secretion; but there is a small quantity of fluid containing mucus, no acid, and no traces of the mother substances of the ferments. After a test-breakfast, during the pre-atrophic stage, less than the normal quantity of contents is obtained, thick and ropy, and of high specific gravity; the bread is for the most part unchanged; occasionally the mucus is tinged with blood. There is always less fluid in the contents than normal, for secretion (except mucus) is less in quantity, and the motor function is active. There are no organic acids, or mere traces (except where butyric fermentation is active but transient and accidental), no free HCl, and combined HCl may also be completely absent, and the reaction of the contents may be neutral, or even alkaline. The small quantity of HCl secreted is combined with inorganic bases, or is neutralized by the alkaline exudate from the blood-vessels. The acidulated tube digestions show in the beginning diminution of the pepsin, and the labferment and the labzymogen are also less active than in the normal contents. The ferments, however, may not be so deficient as we should expect from the very small free hydrochloric acidity, and they show the destruction or degeneration of the peptic glands with greater exactness than does the acid secretion. There is no excessive or characteristic germ growth, and the tube fermentation tests are negative. After the test-meal of Germain Sée, less than the normal quantity of contents is obtained, the food being but slightly changed. There is no peptone, and the propeptones and syntonin are present in very small quantity. The tests for achroödextrin and for maltose are positive, ptyalin digestion being active. The contents are thick, for there is less than the normal quantity of fluid in the stomach. The secretory signs display the pathological changes in the mucosa. The excessive secretion of mucus is persistent. The free HCl diminishes and disappears, and the diminution of combined HCl becomes greater and finally disappears. The pepsin diminishes in proportion to the total hydrochloric acid (H + C). But before the chief cells are degenerate, the acid secretion—and with it the secretion of the ferments—may be improved by soothing medication to control the secretion of mucus and to relieve the subacute interstitial gastritis. The improvement is in inverse proportion to the degeneration and destruction of the peptic glands. But mucus is actively secreted even after the peptic glands have been transformed or destroyed.

The motor function is intact, the stomach emptying itself

in the normal time. Expression is easy, unless the contents are very coarse or thick; it is never difficult for the same reason that it is difficult in myasthenia—viz., the unretracted, flabby condition of the stomach. There is no serious disease of the stomach in which the germ growth is less than in chronic asthenic gastritis. But butyric fermentation occurs intermittently in a majority of the cases. In the morning, fasting, the stomach is empty, or contains a small quantity of mucus and saliva. The sediment of the morning contents contains a large quantity of single and of exfoliated masses of cylindrical cells and leukocytes, mixed with mucus. Absorption is diminished in proportion to the diminution of secretion. The functional signs are constant and do not rapidly change, either spontaneously or under the influence of diet or remedies, except in the manner and for the reasons already stated.

The urine presents no characteristic change, but the digestive fall in acidity is less marked than in health, and the acidity of the twenty-four hours' urine is high, and there is often a deposit of urates and of uric acid. In the production of these changes the intestines often play as important a part as does the stomach.

Prognosis.—Chronic asthenic or catarrhal gastritis is a serious disease which may be arrested by proper treatment, and may be completely compensated by healthy intestines. Consequently, the prognosis is dependent largely upon the condition of the intestines and upon the ability of the patient to obtain the proper food. It is always more serious in malarial and warm regions, on account of the frequency of hepatic and intestinal diseases. A perfect cure is possible even after degeneration of the peptic glands has begun. But the greater the proliferation of the cylindrical cells, the greater the degeneration of the peptic cells, the greater the number of beaker and of ciliated cells, the greater the mucous transformation, the greater the diminution of the total hydrochloric acid, of the ferments, and of the quantity of secretion—just so much less will be the probability of the restoration of normal secretion. The examination of a piece of the mucous membrane is an important aid alike in diagnosis and prognosis. But too much weight should not be given to this kind of information, for the gastritis may not be general, nor in the same stage in all parts of the stomach.

Asthenic gastritis may also be complicated by motor insufficiency, but this complication is rare and occurs late, when the muscular layer becomes infiltrated. The terminal period,

with inanition and gastro-intestinal atrophy, is often accompanied by pernicious anemia.

Differential Diagnosis.—Chronic catarrhal or asthenic gastritis is liable to be mistaken for adenasthenia gastrica, neurasthenia gastrica, myasthenia gastrica, and carcinoma.

In a general manner the dynamic affections are excluded by the gastric symptoms of asthenic gastritis being so closely related to the digestive period and to the taking of food, and being excited also by solid food, particularly meats, much more than by fluids. In gastritis the symptoms are more closely connected with the alimentation; in the dynamic affections they are more intimately connected with the state of the nervous system. In gastritis the functional signs are constant: in the dynamic affections the functional signs may be normal, or, if pathological, they may vary from day to day under petty and seemingly inadequate influences. Gastritis is most frequent after, and the dynamic affections before, the thirtieth year.

In adenasthenia gastrica nausea and vomiting do not occur. There may be diminution and even absence of HCl, but there is no excess of mucus, nor active epithelial exfoliation, nor traces of blood in the contents; and the ferments, though often are not always continuously diminished. The functional disorder often begins suddenly, after shock, injury, or moral depression; chronic gastritis develops slowly, or follows the acute disease. In adenasthenia the diet is without influence on the slight local discomfort; in asthenic gastritis the local symptoms are made worse by an improper diet. The treatment suitable to adenasthenia is without benefit in asthenic gastritis, which demands a special diet and local remedies much less excitant.

In neurasthenia gastrica the abdominal symptoms are digestive, and are excited by the simple activity of the stomach, somewhat regardless of the quality of the diet. One article of food is digested little better or worse than another. In neurasthenia gastrica the patient is "dyspeptic" because he suffers and complains, there being no marked and constant chemical insufficiency. The secretory signs are in vivid contrast with the hypochlorhydria, excess of mucus, and constant diminution of the ferments found in asthenic gastritis.

In myasthenia gastrica the signs of the motor insufficiency at once make the differentiation, except in the rare cases where motor insufficiency is a complication of the asthenic gastritis. Fluids are worse borne than a dry solid meal, the contrary being true in gastritis. The chemical signs of myas-

thenia—secretion being rarely constantly diminished—are in contrast with those of gastritis. But asthenic gastritis may develop as a complication of myasthenia; and, apart from the presumption being in favor of the myasthenia as the primary trouble when the two diseases are found associated, only a knowledge of the development of the case could clear up the difficulty.

There is little chance of confounding gastritis with carcinoma, if the case has been well studied. The secretory signs in the two diseases may be alike, for in both they are produced by catarrhal or asthenic gastritis; but there the resemblance ends. There may be motor insufficiency in carcinoma, and this occurs late (terminal period) in asthenic gastritis. Not one sign, but the two groups of signs, in the two diseases, should furnish the reasons for the decision. For the differential diagnosis, see the chapter on Carcinoma.

Treatment.—It is much easier to prevent chronic gastritis than to arrest the disease when it is fully developed. For this reason the functional disorders of the stomach should receive careful treatment, and the stomach should be guarded and favored in all diseases of other organs which play a part in the causation of gastritis. The prophylaxis consists in strict digestive hygiene, a proper diet, and, what is more often disregarded, but is equally important, in the avoidance of all drugs which disturb its functions and injure the mucous membrane. Dietetic abuses have become so firmly fixed in the customs and habits of the people that there is little danger of the disease disappearing, or of its becoming easy to find a healthy stomach more than forty years old. But probably some good may be done by recommending, to those who most need the advice, temperance in eating and drinking, and by correcting gross violations of digestive hygiene.

The treatment of secondary gastritis is in part the proper treatment of the disease which caused it. The management of the disease of the stomach is modified by the necessity of controlling or of removing its cause. This subject is discussed in the section on the Vicious Circles of the Stomach.

The most important indications to be met by the treatment of chronic asthenic gastritis are: (1) To protect and to favor the inflamed organ; (2) to maintain intestinal compensation; (3) to excite the deficient secretion of the stomach by physiological remedies; (4) to remove the excess of mucus when it is thought best to do so; (5) to treat gross symptoms and complications.

The most valuable principle of affording the greatest possible protection to a diseased organ should guide us in the treatment of the anatomical diseases of the stomach. The proper treatment of chronic gastritis is an embodiment of protection and of favoritism. A great deal of injury can be done by trying to force the stomach to do its full chemical work, and by filling the organs with antifermentatives and antiseptics to drive out an imaginary evil.

A diet properly regulated is our most important remedy in asthenic gastritis, and is often capable alone of suppressing the symptoms and of arresting or curing the gastritis. The chemical work of the stomach being very much reduced on account of the diminished secretion, the disintegrating and solvent work of the gastric juice should be as far as possible done by the preparation and the thorough mastication of the food. The motor power of the stomach must be carefully nursed and guarded, for the maintenance of the balance of nutrition depends on the delivery to the intestines of food in sufficient quantity, and capable of digestion and utiliization by the part of the digestive tube below the pylorus. The disease is in the stomach, but the intestines have the most to do with the selection of the diet.

Before and after each meal the mouth and the teeth should be cleaned, and all the food eaten should be perfectly fresh. The intestines being no longer protected by the acid gastric secretion against bacterial invasion, every precaution should be taken to have the food pure and sweet.

The gastric acidity is not sufficient to arrest the action of the ptyalin, and the starches should be most thoroughly chewed and insalivated, and the work of the intestines thus made lighter.

Physical and chemical irritants should be excluded from the diet. Alcohol is particularly injurious, irritating the inflamed mucous membrane, by which it is not actively absorbed as in health, and a large part of it is consequently conveyed into the intestines. The food should contain only a moderate quantity of salt, and should never be highly seasoned. A small quantity of mild sauce may be used to appeal to the palate. Fat in excess, as demonstrated by Leven, is also a gastric irritant, and diminishes motor activity. More than a very small quantity of cane-sugar must be prohibited, as an intestinal prophylactic measure. Milk-sugar is not objectionable if the small intestine is healthy. But all forms of sugar are hydragogue, and in excess and in concentrated solutions are irritant, and unless rapidly absorbed are liable to ferment in

the intestines. It is a rule established by clinical experience that sweets and fats must be reduced to a minimum in the treatment of asthenic gastritis, but moderate quantities of fresh butter should always be given, and are usually well borne.

The sweets may be partly replaced by gelatinous foods, which possess a high force value, resist fermentation, and are rapidly digested and utilized by the intestines. Calf's- and pig's-foot jelly and calf's head (milk sauce) may be utilized for maintaining nutrition.

The albuminous foods escape gastric digestion, but this affords no reason for diminishing the quantity of this class of food. But the quality and the preparation must be favorable to intestinal digestion and utilization. The meats should be tender, lean, finely divided (chopped or scraped), and as free as possible from indigestible tissue (skin, tendon, blood-vessels, fascia). No fried or greasy dishes should be permitted, and all the meats, poultry, and game should be either stewed, broiled, or grilled.

Milk is well borne in some cases, and should then be permitted as a drink and in the preparation of vegetables, sauces, with cereals, etc. It should never be given in the exceptional cases with gastric fermentation, nor where there is stagnation in the small intestine; on the appearance of the first signs of gastric or intestinal fermentation it should be immediately excluded.

Not more than one glass of fluid should be allowed with a meal. The starch-containing foods should be thoroughly masticated and insalivated before being washed down with water or swallowed. No alcoholic drinks should be permitted. Plain water and very weak tea are the most suitable drinks; chocolate and cocoa are better borne than coffee, and in exceptional cases may be permitted, particularly Hauswaldt's "vigor chocolate."

The following articles should form the staple foods, and should be given in combination and quantity in keeping with the principles of dietetics, so as not only to maintain, but, if need be, to improve nutrition. But where a trouble of the intestines is associated with the gastritis, it may be necessary to restrict and to lower the diet temporarily below the requirements of nutrition until the intestinal disease is under control and digestive compensation is reëstablished. All tender lean meats, beef, mutton, calf's-brain, sweet-bread, young chicken, squab, animal jellies, lean and fine-meated fish, soft part of oysters in season, whites of eggs

cooked just enough to hold together, and the yolks of eggs (when there is no intestinal putrefaction), and milk, when well borne (which is not the case if there is intestinal fermentation); preparations of rice and wheat, thoroughly cooked, spinach passed through a sieve, cooked tender sprigs of celery, mashed French peas, tender string beans passed through a colander, and tender sprigs of lettuce with a little salt, white bread toasted through and through, or the crust of roll; and in some cases thoroughly cooked mashed potatoes or a mealy boiled potato may be permitted.

All other articles are prohibited until the disease is arrested, as indicated by the functional signs, and until digestive compensation is well established. But the deficient chemical work of the stomach will necessitate the observance of hygiene, and the continuance of a diet favoring and nursing the motor power of the stomach, and maintaining strict intestinal prophylaxis. It may be necessary to prescribe a fluid diet for a short time, to give four or five small meals a day, and to permit only such foods as are evacuated by the normal stomach in about three hours. Select, in the beginning of treatment, such foods as leave the stomach rapidly, such as have little action on its mucous membrane, and such as are utilized by the intestines in sufficient quantity to support or to improve nutrition.

This is the rule when it is necessary to protect and favor the stomach; but such is not always the proper course. The diet may be used as a physiological remedy to excite secretion when excitation is desirable. The meats are strong excitants of secretion; the green vegetables, fruits, and sweets act in a similar manner; and peptones and albumoses may be prescribed for the same purpose, and condiments may be permitted in moderation. It is better to use food as an excitant remedy than to prescribe drugs for the same purpose; but the intragastric douche with a physiological salt solution is a valuable synergist.

The bowels should be kept regulated by massage, electricity, and injections to which glycerin may be added. If intestinal fermentation becomes active, a dose of calomel should occasionally be given.

The appetite may be so diminished as to require special treatment, the increase of the appetite improving also the psychic state of the patient. An infusion of condurango or calumba may be given with a tablet of strychnin.

The general restorative and hygienic remedies—physiological living, hydrotherapy, electricity, etc.—should not

be neglected, and every means should be used to maintain and to improve nutrition, to tone and to rest the nervous system, to protect and to favor the stomach, and to secure digestive compensation by the intestines. Stomach washing may be employed to remove the very large quantity of mucus, but we rarely employ it in asthenic gastritis unless there is butyric fermentation. Lavage should be done at bedtime or in the morning before breakfast, using plain warm water, or a little lime-water (1 to 10) may be added. If the patient is not accustomed to the tube, a glass of hot water may be slowly sipped an hour before breakfast. The addition of a few grains of sodium chlorid and calcium chlorid to the hot water usually controls the butyric fermentation. We rarely prescribe mineral waters and so-called mucus-solvents in asthenic gastritis. Indeed, the good motor function and the traversing food and water, aided, if need be, by lavage, are sufficient to remove the excess of mucus; and it would seem wise to leave enough sweet mucus on the surface of the stomach to protect the mucosa.. The long preservation of the glandular division of the mucosa in health and repose is due to the non-injurious and protecting coat of thick mucus. Some aid may be rendered the intestines in their digestive work by supplementary chemical treatment, using hydrochloric acid, and, in the advanced stage of the destructive process, also pepsin, or papoid alone. When the hydrochloric acid is used as a digestive agent, two doses of the dilute acid (about 20 drops) should be given during the functional activity of the stomach, about sixty and ninety minutes after the beginning of the meal. Pepsin should always be combined with it. In the few cases with gastric fermentation, a dose of the acid may be given as an antizymotic, half an hour before each meal. Papoid may be employed without the acid when the stomach is atrophied or when a complicating nephritis exists. But supplementary chemical aid should never be employed until the period has arrived for excitant treatment, for, while it may aid the intestines, it may injure the stomach.

II. GASTRITIS GLANDULARIS PROLIFERANS, OR CHRONIC HYPERSTHENIC GASTRITIS.

Pathological Anatomy.—In chronic asthenic gastritis the cylindrical cells which cover the surface of the mucous membrane and line the mucous ends of the ducts of the glands proliferate, and may replace the degenerate chief and border

cells, thus converting many of the peptic into mucous glands. Or the fundus of the degenerate gland disappears and nothing is left but the elongated duct of the gland, lined with cylindrical cells, with beaker cells, and with ciliated columnar epithelium. In this process the proliferation and infiltration of the connective tissue may play a more or less important part. In chronic proliferating glandular gastritis the pathological process is different and in bold contrast. There may be the same infiltration and proliferation of the connective tissue, and



Fig. 22.—Gastritis glandularis proliferans. × 240. (Authors' specimen.)

the same wandering of leukocytes, which are, as a rule, oxyphile. But the proliferation affects the cells of the peptic glands; the cylindrical mucus-secreting cells remain normal, or degenerate and disappear over large areas of the surface of the mucous membrane, and many of the pyloric glands become lined with cells resembling the chief and border cells of the peptic glands (Fig. 22).

The mucous membrane is covered with mucus, which is stained with blood and is ordinarily thin and mixed with the nuclei of partly digested cells. The surface of the mucous

membrane is very vulnerable; it is not, ordinarily, mammellated, but the little villosities may be prominent, and the mucous membrane is firm and thick. Large areas of the mucous membrane are denuded of epithelium, and a peptic (round) ulcer is not rare. The surface epithelium is usually destroyed by postmortem digestion, but it may be found well preserved in small pieces of the mucous membrane obtained through the tube; there are no beaker cells, no reverted ciliated columnar epithelia, no mucoid degeneration of the cylindrical cells, but rarely a very few cylindrical cells may show mitosis (normal). In old cases the superficial layer of the mucosa may be richly infiltrated, and the nutrition of the surface epithelia may be cut off and their reproduction arrested. The positive characteristic of the gastritis is the proliferation of the border and the chief cells. Some cases show few border cells, and some few chief cells, and the predominance of the one or the other cell may depend on the functional state of the gland when the specimen is obtained—in the morning, when the stomach is in repose and has been empty for some time, or during digestion, or during the period when the stomach still has stagnant or retained food in it. But the glands are ordinarily in a state of functional activity, and the chief cells greatly diminish in number. It is possible that autodigestion and their normally low vitality have something to do with their disappearance. Under the microscope the glands appear lined with cells which are well preserved and closely packed, and some of the younger ones stain intensely. The lumen of the glands is filled with granular matter, a few leukocytes, and the remains of chief cells. Many of the cells contain more than one nucleus. The swelling of the mucosa is due to the active proliferation of connective tissue in the first anatomical stage of many cases, but the inflammation may be interstitial in the beginning, or the interstitial inflammation may develop in the course of the evolution of the case. Proliferating glandular gastritis rarely ends in anadenia gastrica, though many of the glands may be compressed and deformed, and even destroyed, by the interstitial inflammation, and in old cases many of the cells undergo granular and mucoid degeneration and vacuolation. Some of the pyloric glands contain a few border cells, and these glands, as Hayem first discovered, may be converted into true peptic glands. The rich and excessive secretion is the natural sequence of the pathological alterations of the glandular layer.

Clinical Description.—The symptoms of hypersthenic or proliferating glandular gastritis may be confined to the nor

mal digestive period, and they cease entirely after the stomach has emptied itself unless secretion is continuous. Like chronic asthenic gastritis, the disease may be perfectly latent for a long period; or it may manifest itself by distant, and not by local, subjective symptoms.

The appetite is good, and often greatly increased, but there is no selective desire for spicy articles or for sour food. Soon after the meal the stomach may feel full, and there is for a short time a sense of well-being; but as digestion proceeds, heartburn, eructations, and pain begin. Thirst is usually strong, and the pain and the burning are relieved by drinking water. There is no nausea and no vomiting. The local pain increases with the evolution of digestion, but is relieved by fluids, albuminous foods, and alkalies. The intensity of the symptoms is proportionate to the physiological action of the food on secretion, to the state of the nervous system, to the activity and richness of secretion, and to the time during which it remains undigested in the stomach. Starches, sweets, and fats are not so well borne as is albuminous food, and a glass of milk may be digested with but little discomfort. This is the simplest and the mildest type of chronic hypersthenic gastritis, which may have begun suddenly with an attack of acute gastritis, or have been preceded, for a variable period of a few months or years, by adenohypersthenia, or which may have long remained latent or manifested itself intermittently by subjective symptoms.

Where acetic or butyric fermentation is associated with the glandular gastritis and morbid sensibility, the preceding symptoms are intensified; and during the period when, normally, digestion would be in the stage of decline, the pain may become paroxysmal, and radiate from the stomach over the abdomen and into the back. The symptoms are incompletely relieved by water, by albuminous foods, and by alkalies. There is often headache and nausea, which may terminate with vomiting. But the symptoms maintain the same relation to the evolution of digestion, and to the quantity and composition of the diet, as in the simple form.

In other cases the symptoms are not confined to the normal digestive period, but encroach upon the period of gastric repose. The local symptoms then become more continuous, with digestive exacerbations, and with nocturnal attacks of pain and nausea, and sometimes vomiting. The glandular gastritis is complicated with prolonged digestion produced by supersecretion. Stagnation may end in retention, and the stomach may never completely evacuate through the pylorus

the food taken into it; or secretion may become continuous, and the organ never get rest from irritation.

The simple type with digestive symptoms, at any moment, through dietetic errors or through fatigue, may become marked by periods when fermentation is active; or stagnation or retention may occur for a few days. The progress of the disease is then characterized by periods during which all the symptoms are exaggerated and accompanied by nausea and vomiting, lasting a number of days; and by periods lasting a week or a month or longer, during which the patient is comparatively comfortable.

The emaciation becomes greater and greater as the suffering, the loss by vomiting, the destruction of food by fermentation, and its waste in intestinal putrefaction increase. The disease now becomes a menace to life. Continuous secretion and retention, with fermentation, excite more and more the inflammation, which becomes more and more interstitial and the destruction of the glands and the muscular fibers begins. The patient, who is generally a man between twenty and forty, is now emaciated, sallow, with long suffering traced in all his features. The appetite is irregular and sharp, and in order to satisfy it food is taken irregularly and frequently. As the rule, the morning is the most comfortable part of the day, but in the afternoon the accumulated contents—consisting of the foods and fluids ingested, the secretions, and the products of fermentation and digestion—may so irritate the mucous membrane as to produce a severe paroxysm of pain, nausea, and vomiting. The vomit consists of a greenish or a grayish fluid, mixed with undigested starch, ropy, acid, and separating into three layers on standing. The dinner is taken with appetite, and from four to six hours later a second paroxysm, like that of the afternoon, may rob the patient of all but a few hours' sleep. There may be only one paroxysm during the twenty-four hours, and the vomiting may be replaced by diarrhea. Ulcer may develop at any stage of the evolution of the disease, from the simple digestive form to its terminal period—supersecretion, stagnation, fermentation, and finally retention being the intercurrent episodes.

The stage of hypersthenic gastritis characterized by continuous secretion and motor insufficiency constitutes a symptom-group sometimes described as Reichmann's disease. The same symptom-group may characterize a stage in the evolution of myasthenia, of gastroptosis, and of pyloric obstruction.

Symptomatology.—The appetite in the hypersthenic form is well preserved, and although often diminished during the

paroxysms of pain and vomiting, it is never completely lost, as may be the case in anorexia nervosa, in cancer, and in asthenic gastritis, and in myasthenia with retention and fermentation. On the other hand, during the periods of improvement the appetite is unusually sharp, and may become as imperative as in bulimia.

Thirst is also a common symptom, and may become very strong during the height of digestion and after profuse vomiting and diarrhea. The patient soon learns that the pain is moderated by drinking water, and in the beginning of paroxysms may take it in very large quantities.

Pain is a characteristic and frequent symptom, and varies in intensity and in its qualities in the different stages. So long as the motor power is normal and there is no fermentation the pain is digestive, and is most intense when the quantity of free hydrochloric acid is greatest. But in this stage of the disease, after a small non-irritating meal, it may be replaced by discomfort and heartburn. When motor insufficiency appears as a complication, the pain recurs in paroxysms, due to the accumulation of the irritant contents. It is located in the epigastrium, and radiates to the left and over the abdomen and into the back, burning and unbearable. It is relieved by albuminous foods, such as meats, milk, and eggs, and, more completely, by large doses of alkalies. It subsides also after vomiting (if complete) and lavage and after the evacuation of the stomach into the duodenum.

Nutrition in the beginning is well preserved, but emaciation develops with the increase of fermentation, of vomiting, of retention, and of disturbance of the functions of the intestines. In the advanced stages it may become extreme, and may be associated with cachexia as pronounced as in cancer.

The physical signs are not characteristic, and reveal a complication—such as ulcer, stagnation, and retention—more often than they reveal the gastritis itself.

The functional and bacteriological signs not only aid in making the diagnosis, but also indicate the stage which the disease has attained in its evolution, and are valuable guides in the treatment. As regards the functional signs, the disease may be divided into three stages or periods: (1) Initial period; (2) period of prolonged digestion; (3) terminal period.

During the *initial period* the motor function is sufficient, and the stomach empties itself in the normal time. The resting stomach contains no remains of undigested food nor digestive products. The tube, introduced in the morning before break-

fast, withdraws possibly a few cubic centimeters of fluid, slightly acid, and containing mucus, and sometimes saliva; the sediment contains spiral cells, nuclei, sometimes a few wandering leukocytes, bacteria, but no yeast or sarcinæ. After the test-breakfast about the normal quantity of contents is obtained, grayish or greenish, with a peculiar acid odor. The bread (except starch) is well digested, but the mixture is not so homogeneous as in health, and contains an excess of mucus. There are no organic acids. The total hydrochloric acidity is very high (70 to 120), of which part (C) is combined (10 to 40) and part (H) is free (20 to 60). The tube digestions are very active, the acidulated 50 per cent. dilution digesting as rapidly as the undiluted filtrate in health. The labferment and the labzymogen are both very active. The contents obtained two hours after the test-meal of Germain Sée contain nearly all the meat in solution, only a few fibers already undergoing disintegration being discoverable with the microscope. Syntonin and propeptones are abundant, and both the biuret and Almèn's reactions for peptones are plain. The starch is not digested as well as in health, and there is no accumulation of ptyalin products. The tube fermentations are negative, and the total quantity of the contents obtained after the test-meals, as estimated by the method of Mathieu or of Strauss, is sometimes normal, but is ordinarily excessive, on account of the usually excessive activity of secretion. The hydrochloric acidity (H + C) is excessive, and the excess may be due to an excess of both (H) and (C), or to an excess of (H) chiefly, or of (C) chiefly. The free (H) hydrochloric acid may appear too early in the evolution of digestion (before thirty minutes), and the acme of hydrochloric activity may occur during the second hour of the digestion of the test-breakfast. The line which represents the evolution of (H + C) displays no sudden rises and falls, although it runs abnormally high. Secretion ceases with the evacuation of the stomach, which occurs within the normal time.

During the *period with prolonged digestion*, two conditions may be found. In the one, the signs are the same as in the initial stage, with a few differences. The contents are excessively rich in (H + C) hydrochloric acid and the ferments. The albumin digestion is rapid, and starch digestion is diminished. The evolution of secretion is exposed to the same disorders. But the quantity of the contents is greater than normal, and the excessive quantity is due to supersecretion or to spasm of the pylorus. The stomach contains remnants of the roll, and products of pepsin digestion. After the acme

of digestion too much free HCl is always found, and, as digestion proceeds toward its termination, the percentage of free hydrochloric acid increases and that of the combined hydrochloric acid diminishes. The stomach may contain some of the test-breakfast at the end of three hours. In the ordinary course of events the stomach may succeed in emptying itself between each meal, or the digestion of one meal may not be completed when another is begun. But when the prolongation of digestion is due to supersecretion, the stomach always empties itself during the night unless secretion is continuous. The stomach is empty one and one-half hours after the water test, or it contains a variable quantity of secretion, rich in acid and ferments. There is no myasthenia; digestion is prolonged because secretion is excessive, and secretion ceases soon after the stomach is empty. The second condition is characterized by continuous secretion. The signs are nearly the same as in the first form with supersecretion, but the tube, introduced in the morning, before breakfast, withdraws a greenish or grayish ropy fluid, without or with alimentary residue, varying from 50 to 250 c.c. or more in quantity, containing free hydrochloric acid (5 to 20 or 50), possibly a small quantity of combined HCl (digested cellular protoplasm), digesting albumin actively in the tubes, and possessing a sp. gr. of 1004 to 1006. About the same quantity of secretion may be obtained after the stomach has been washed out the previous evening, nothing (food, water, nor saliva, etc.) having been ingested or swallowed during the night. The glands continue to secrete throughout the twenty-four hours. In continuous secretion the starches undergo no change. There may be slight fermentation—a small quantity of organic acids in the contents, a moderate amount of budding yeast, sarcinæ, bacteria, particularly cocci, and, possibly, a little gas formation in the fermentation tubes.

Secretion during the *terminal period* may remain excessively rich and be less than normal in quantity. The evacuation of the stomach is then abnormally rapid, unless the motor function is made insufficient by a complication. Or, again, the hydrochloric acidity (H + C) may be less than in health, and secretion may be about normal in quantity, or it may be excessive, or it may be continuous. Consequently, digestion may be finished too rapidly, in the normal time, or it may be prolonged. But the prolongation is the result of the excessive secretion and is not due to motor insufficiency. The secretory signs reveal the progress of glandular destruction by compression. There is, however, a possibility of being

badly deceived in this matter, for secretion may be reduced in quantity and in richness at any period of the evolution of chronic proliferating glandular gastritis by acute interstitial gastritis. But the suppressed hyperchlorhydria will reappear if the complicating acute condition is relieved by protecting the stomach against all irritation and by favoring it in its work or by giving it functional repose for a few days. Sedation and rest produce—or, better, restore—excessive glandular activity, and this sequence occurs in no other disease of the stomach.

Hypersthenic gastritis may be complicated by myasthenia, by pyloric obstruction, and, possibly, by motor insufficiency due to infiltration of the muscular layer. Great difficulty may be experienced in distinguishing these complicated cases with stagnation or retention from the simple cases of hypersthenic gastritis with prolonged digestion.

In the contents, or in the vomit, or in the wash-water, signs of gastritis may be found—blood, altered cylindrical cells, leukocytes or their nuclei, and sometimes small pieces of the vulnerable mucous membrane. The pieces of the mucous membrane show the nature and characters of the pathological process in evolution at the point from which they come, which is nearly always the same as the process which predominates in other parts of the stomach. But gastritis is not always of the same character and stage and intensity at all points of the mucosa.

The urine easily precipitates the earthy phosphates, but the total quantity of phosphoric acid eliminated in the twenty-four hours may be above or below the normal. The total quantity may reach as high as five gm. or may fall as low as one or two gm., the average elimination being 2½ gm. in the twenty-four hours. But, be the total quantity large or small, the urine, as a rule, is cloudy when voided, and the earthy phosphates are precipitated by heat because the urine is nearly neutral or alkaline. The diminution of the acidity of the urine is proportionate to the amount of acids found in the stomach. The sum total of the acidity of the urine and of the gastric juice in health is a constant quantity, and this inverse relation is only disturbed in disease by fermentation and by increased or diminished secretory activity, chiefly of the intestines and its annexed glands and of the skin. In hypersthenic gastritis the acidity of the urine is markedly diminished during digestion; and when digestion is prolonged by excessive or by continuous secretion, the reaction of the urine passed during the twenty-four hours is nearly neutral or alkaline, and it

may be milky with precipitated phosphates. The quantity of urea is increased and the increase is often absolute. This is due in part to the excessively albuminous diet, to the increased alkalinity of the blood, and, probably, to the excessive formation of peptones. The increased relative percentage of urea is due to the concentration of the urine by diminished absorption or by increased loss of water by the stomach and intestines in vomiting, retention, lavage, and diarrhea.

The chlorids are constantly and absolutely diminished in proportion to the vomiting, retention, lavage, and activity of gastric secretion. The urine formation, being continuous, shows at each moment the chlorid percentage of the blood. The blood, during the excessive gastric secretion, is poorer than normal in chlorids. Consequently, the excessive quantity of chlorin removed from the blood by the stomach would, without any other disturbance, diminish the total quantity of chlorids eliminated in the urine during the twenty-four hours, and, to a greater degree, during gastric secretion.

Diagnosis.—The slow evolution, the subjective and the objective signs of hypersthenic gastritis, are so characteristic that the diagnosis is not often difficult. The symptom-group presented at any stage would fix the mind on hypersthenic gastritis as one of the probabilities. The differential diagnosis may be a hard problem.

Differential Diagnosis.—Hypersthenic gastritis may be primary, or it may be a complication developing in the orderly evolution of other diseases of the stomach, such as myasthenia, displacements, ulcer, pyloric obstruction, and, seldom, carcinoma. Without the functional and bacteriological signs, and careful attention to the evolution of the case, the differentiation is only a lucky or an unlucky guess at the truth. If the case be incompletely studied, carcinoma may be easily confounded with hypersthenic gastritis. Pain, slow evacuation of the food, emaciation, and cachexia are symptoms common to the two diseases. But a comparison of the distinctive features of the pain would place in our hands the guiding thread which would lead to a correct conclusion. It is useless to recall vague and slight variations when the functional and the bacteriological signs are so widely different in the two diseases. The excessive hydrochloric acid in the one, and its diminution (little or no H, little or no C) in the other; the active and rapid albumin digestion in the one, and its slowness, and incompleteness, and possible absence in the other; the absence of lactic acid fermentation in the one, and its frequent

active formation, after stringent precautions, in the other; the almost constant character of the germ growth in the one, and its rapid changes and special character, in keeping with the quick changes in the chemical properties of the more and more stagnant contents of the other—are so many signs which place the two diseases in vivid contrast. The appetite, the effects of food, the urine, the blood, and the evolution, present other important differences. Very rarely carcinoma may be engrafted on an old ulcer, and the differentiation may be exceedingly difficult, and may demand a close and complete study of every little symptom and sign.

Myasthenia may become accompanied by fermentation, which might produce secretory irritation, and present a symptom-group closely resembling the stage of hypersthenic gastritis associated with prolonged digestion and fermentation. Or the stagnation of the food in myasthenia might produce supersecretion. A knowledge of the evolution of the case up to the moment of examination would clear up the difficulty and reveal the primitive character of the myasthenia or of the gastritis. The therapeutic test gives valuable information. If the stagnation-caused fermentation in myasthenia is controlled by diet and by lavage, and if the treatment suitable to myasthenia is adopted, the excessive and rich secretion due to irritation rapidly subsides; but hydrochloric superacidity would continue unabated during the prolonged digestive period in hypersthenic gastritis. The anatomical signs of the gastritis would be absent in the particular stage of myasthenia under discussion. Myasthenia and hypersthenic gastritis resemble each other only when both are accompanied by delayed evacuation of the food and by hydrochloric superacidity. The existence of continuous secretion excludes simple myasthenia, and the hydrochloric superacidity of myasthenia is quickly relieved by protecting the stomach from irritation and by giving it functional rest. The lines representing the evolution of secretion in myasthenia show sudden rises and falls. The water test gives different results in the two diseases, for myasthenia is accompanied by stagnation of liquids due to motor insufficiency, and hypersthenic gastritis may be accompanied by prolonged digestion due to supersecretion. Moreover, in myasthenia expression is difficult, the stomach is flabby, loses its form, does not retract as it becomes empty, and splashes whenever it contains fluid and gas. Contrary signs are present in hypersthenic gastritis with prolonged digestion. For other differential signs, see Myasthenia. But myasthenia may become complicated by

hypersthenic gastritis, and nothing except a knowledge of the development of the case could make the differential diagnosis. The link belongs to either of the two chains, and the treatment is practically the same when both diseases are present.

Pyloric obstruction, gastroptosis, and vertical displacement of the stomach may be complicated by hypersthenic gastritis, and the history may give the primary trouble; the gastritis is most frequent in men and the displacement of the stomach in women. For the differential signs, see Myasthenia, Displacements of the Stomach, and Obstruction of the Pylorus.

Treatment.—The treatment is not the same in all the stages of hypersthenic gastritis, and each of the periods characterized by special functional signs presents particular indications to be met.

1. **The Initial Period.**—The disease gathers more and more force and violence as it progresses, and each day the lesions become more and more extensive and incurable; and hence comes the stringent necessity for correct and consistent treatment during the initial period.

No more striking instance can be found in pathology of the danger of neglecting a disorder of the stomach and of not curing it while in its early stage. The prophylactic treatment—so important on account of the part which every disease of the stomach plays in pathogenesis—should not be neglected. Prophylaxis is particularly important in the hypersthenic diseases, for they possess a strong inherent tendency to extend and to progress. After the initial period is established the indications are: (*a*) To protect the mucous membrane against all forms of irritation; (*b*) to maintain the balance of nutrition by a suitable diet; (*c*) to control or utilize the excessive secretion; (*d*) to treat the gross symptoms; (*e*) to prevent stagnation or retention.

The mucous membrane may be irritated by drugs, by the diet, and by the excessive hydrochloric acidity. Tonics, nervines, purgatives, and all drugs which act as local irritants and excitants of secretion should be avoided; indeed, not only avoided, but absolutely prohibited.

The physiological action of the diet should be indifferent, non-excitant—no condiments, spices, vinegar, highly seasoned sauces, fermenting or decomposing foods, or alcoholic drinks. Such foods and drinks should be selected as do not excessively excite secretion, and which utilize the secretion which is formed. Consequently, all articles of food which in

health leave during their digestion a noteworthy quantity of hydrochloric acid free, should be prohibited. The excessive hydrochloric acidity of the contents irritates the mucous membrane, which in hypersthenic gastritis may be particularly sensitive to the action of free hydrochloric acid. The diet is selected by the needs of nutrition, by the functional activity of the stomach, by the imperative necessity of avoiding secretory irritation, and by the state and functional power of the intestines. The diet should also be so regulated as not to tax the motor function of the stomach too heavily, the development of motor insufficiency rendering the disease much more serious and dangerous.

During the initial period nutrition is commonly well preserved, if the good appetite has been heeded. If emaciation exists during this period it is the result not of the disease, but of faulty alimentation, or possibly of a too free use of purgatives, or of intestinal disease. Consequently, the demands of nutrition are met by maintaining its equilibrium. An exclusive weakening diet is improper, and it is dangerous to weaken by inanition the voluntary and the involuntary muscles, thus favoring myasthenia and prolapse of the stomach, diminution of abdominal tension, intestinal stasis, and inequality of the circulation. Guided by the active peptonization which is one of the functional characteristics of this disease, the mistake is often committed of prescribing an exclusively albuminous diet incapable of maintaining the balance of nutrition, except possibly at a very low level. The balance of nutrition can be maintained and the functional power of the stomach respected by a diminution and a careful selection of the carbohydrates and fats in combination with an increased quantity of proper albuminous foods.

Physiologically, the meats are powerful excitants of secretion, and their digestive products are even more active, and the red meats require a long time for their digestion. Consequently the meats are not ideal foods in hypersthenic gastritis; but they are of great nutritive value and combine large quantities of HCl. The meats and the other albuminous foods which are evacuated rapidly by the normal stomach should be preferred. In the initial period there is no objection to milk, which is very soothing in its action on the mucous membrane, which combines the free HCl rapidly, and which leaves the stomach quickly. If milk is suitable to the intestines it is wise to begin the treatment with rest in bed, a milk diet for a few days, gradually adding eggs, "vigor chocolate," stewed or broiled young chicken, squab, lean and

fresh fish, and cereals thoroughly cooked and finely ground or mashed after cooking and eaten with milk or with cream, or with broth or milk soup. Rice, the preparations of wheat, oatmeal, hominy, and cornmeal mush are all suitable cereals. The red meats may next be permitted, but they should be free from fat and from fibrous tissue, finely divided, properly hung, and fresh. Next come the purées of green vegetables, mashed starchy vegetables, fruit, sweets, and salads, in the order named. But the intestines may force an exclusion of milk from the diet, and the prescription of a diet almost exclusively albuminous (fermentation), or a diet composed of cereal-thickened soups, meat juice, cereals, milk-sugar, and possibly green vegetables and fruit (putrefaction). Butter should be given in moderation, and always fresh, and preferably unsalted. Whatever diet is prescribed, its action on secretion and on the motor function should be watched, and it should not cause intestinal putrefaction or fermentation.

With each meal—composed of food which is finely divided, contains no chemical or mechanical irritant, is nutritious in small bulk, combines large quantities of HCl, and does not excite secretion (in the normal stomach) more than it requires for its digestion, which leaves the stomach rapidly, and is sufficient in quality and composition to maintain the balance of nutrition —there should be given one or two glasses of fluid, either plain water, or a slightly alkaline water, or milk. A dry diet is very injurious, the water not only serving as a diluent, diminishing the physiological action of the contents on secretion, but also promoting the evacuation of the fluid contents into the duodenum. The prolonged sojourn of the food in the stomach is a disadvantage, exciting and prolonging secretion unduly, and predisposing to the development of continuous secretion, and to the prolongation of digestion through excessive secretion.

The hyperchlorhydria will be greatly influenced by the protection of the mucous membrane against avoidable irritation. The condition of the nervous system may also be a causative factor in the production of the hyperchlorhydria and every means should then be employed to enforce mental and moral repose. The usual remedies for toning the nervous system should be prescribed, one of the most powerful being the Scottish douche. Gastrospinal and cervicogastric sedative galvanization may be given a trial.

To control the excessively acid secretion no remedies are more efficient than the alkaline-sulphate waters, of which Carlsbad water is the type. The many Carlsbad springs vary

but little in the quantity of sodium sulphate, of sodium bicarbonate, and of salt which they contain, the chief differences being in the temperature of the water as it comes from the various springs. Some claim advantages for the natural water or natural salts, but this superiority of the natural over the artificial product may be doubted, although we sometimes employ the "natural" Sprudel salts. The quantity of salt (NaCl) in the Carlsbad water is objectionable, and it is the large quantity of this ingredient in the Saratoga waters which makes them injurious in the treatment of hypersthenic gastritis. The salt either excites secretion (in small doses and weak solutions), or it produces a diminution of HCl secretion by causing acute gastric catarrh. But in small quantity and in combination with an alkali it promotes peristalsis and possesses some other advantages. We ordinarily prescribe artificial salts in the proportions:

Sodium sulphate,	50	50
Sodium bicarbonate,	40	or 20
Sodium chlorid,	10	5.

The first proportion (50:40:10) is more strongly antacid, and the second (50:20:5) acts more decisively on the bowels. A level teaspoonful of the mixed salts should be taken, by sips, dissolved in a glass of hot water, one hour before breakfast. The dose should be increased (proportionate increase of hot water) or decreased until only one full soft movement of the bowels results. The cure should be continued for three to four weeks, the patient taking but little exercise. The Carlsbad cure should not be ordered for the old and the weak, or for patients with valvular heart disease.

A very valuable remedy, both to influence secretion and to allay the excessive irritability and the morbid sensibility of the mucous membrane, is the nitrate of silver (1 : 2000) douche, employed once a week. Or a tablespoonful of a solution of one grain to two ounces of distilled water may be given every morning on an empty stomach. Theoretically, belladonna, on account of its inhibitory action on secretion, and its diminution of reflexes, is indicated; and, practically, it is of very great value when given in small doses ($\frac{1}{50}$ to $\frac{1}{20}$ of a gr.) before meals. Subnitrate of bismuth is very valuable when given in the large doses and manner recommended by Fleiner. Small doses of ergot control the excessive flow of blood to the mucous membrane during digestion.

Pain, vomiting, and constipation may require special atten-

tion. To relieve the pain, no remedy is so effective as the aqueous extract of opium, which may be combined with a good extract of cannabis indica and extract of belladonna, $\frac{1}{10}$ of a grain of each being employed. Codein is not so efficient, but does not increase the constipation. These anodynes should only be used when alkalies, diet, rest, the warm compress, belladonna, ergot, Carlsbad salts, nitrate of silver, and bismuth fail to give relief. It is useless to treat the vomiting with a symptomatic remedy, the act being conservative and protective. Better encourage it by giving lukewarm water, or wash out the stomach.

In addition to the remedies directed against the conditions which underlie it, during the periodical attacks of gastric intolerance a Winternitz or Priessnitz compress should be placed on the stomach half an hour before the meal, and kept there during the period of gastric digestion; and in all cases the compress should be worn at night. The constipation is commonly relieved by the prescribed mineral water, but the bowels may exceptionally require evacuation by a warm water enema, to which a teaspoonful of neutral glycerin may be added, or by gluten or glycerin suppositories.

2. During the **second period** digestion is prolonged. The evacuation of the stomach is delayed, and with the delayed evacuation is associated excessive or continuous secretion, and sometimes fermentation. The remedies used in the first period may also now be useful, but the excessive or continuous secretion must be relieved and the diet changed, so as to avoid producing or increasing the fermentation, and to make the demand on the motor function as light as possible.

Milk must be removed from the diet list and the butter reduced to a minimum quantity. The fermentation may be controlled by an exclusive meat diet for twenty-four or forty-eight hours; but the fermentation will begin anew with the addition of the carbohydrates, provided the long period of gastric digestion has not been shortened. But often with this temporary change in the diet, combined with rest, hydrotherapy, massage, and gastrospinal galvanization, the fermentation may be controlled. A glass of hot water should be slowly sipped every morning an hour before breakfast, during the intervals when the Carlsbad cure is not in progress. This soothing and cleansing remedy is far preferable to stomach washing, as long as the morning fasting stomach is empty and there is no fermentation. The diet during this stage must not be reduced below the needs of nutrition, and

during the temporary employment of an exclusive diet to aid in suppressing the fermentation a nutrient enema should be given daily. Stomach washing is more beneficial than in the initial period, and it may be employed before the evening meal, or at bedtime, or in the morning before breakfast, as there may be, respectively, continuous secretion, or excessive secretion, or continuous secretion and fermentation. Whatever diet be adopted, it is absolutely necessary that the stomach should be empty when a meal is eaten. Consequently it may be best to allow only two meals a day, separated by a long interval, and to use the tube twice a day when secretion is prolonged and does not cease with the evacuation of the stomach, or when secretion is continuous. If the supersecretion is exclusively digestive, it may be best to give a liquid and non-excitant diet, the frequent small meals being separated by intervals long enough to permit the evacuation of the stomach. If secretion is continuous, rectal feeding is the best remedy. All forms of excessive secretion are benefited by belladonna, ergot, nitrate of silver, and bismuth.

3. During the **terminal period** the treatment embodies the same general principles which have regulated the plan of medication in the other stages. The gastric secretion may not be excessively rich, and the diet may be made more liberal. Interstitial inflammation is the new factor with which we have to deal, and it may require, for the relief of its acute exacerbations, even more careful protection of the mucous membrane against irritation, and temporary but complete functional repose. It may be necessary to confine the patient to bed, and to employ rectal feeding for a few days. When secretion becomes diminished in quantity and richness, it may be advisable to douche the interior of the stomach with a physiological salt solution, or to administer five grains of salt in a glass of hot water before breakfast, adding enough sodium sulphate to regulate the bowels; for the intestines demand the same solicitude and watching as in the other stages of chronic hypersthenic gastritis.

III. GASTRITIS GLANDULARIS ATROPHICANS, OR ATROPHY OF THE GASTRIC GLANDS.

Complete primary atrophy or degeneration of the secreting structure of the stomach is not a frequent disease. It does not embrace all cases in which there is prolonged absence of

gastric secretion, for this condition may occur in severe adenasthenia gastrica complicated by interstitial inflammation, and as a termination of other forms of benign and of malignant gastritis. It does not embrace all cases of anadenia ventriculi. The pathological process is atrophic—a progressive parenchymatous degeneration.

Glandular atrophy may be secondary. The severe infectious and the chronic diseases which run a very long course and are accompanied by great emaciation and loss of strength, produce more or less parenchymatous degeneration of the glandular membrane of the stomach, as do also cancer of the breast, of the uterus, and of the intestines. This result is not constant, but is common in the advanced stages of malignant neoplasms of other organs than the stomach.

Diseases of the stomach itself may destroy its glandular membrane. This is specially true of carcinoma of the stomach. The destruction is either direct, the secreting cells not being regenerated, or indirect, the glands being destroyed by compression and by cellular degeneration.

But atrophy of the gastric glands may be primary and not due to any other local or distant disease, or it may represent the terminal period of other forms of gastritis. It is frequent in the marasmus of old age, but is then rarely complete. Chronic inanition may also produce it, and typical forms result from phosphorus-poisoning and from arteriosclerosis. It may be due also to the chronic toxemia of nutritive troubles, or, at least, for unknown reasons, the parenchyma of organs at times degenerates and dies instead of becoming inflamed. The mucous membrane of the stomach is not exempt from this sort of degeneration, and, in spite of its usual great resistance, exceptionally and more markedly and sometimes exclusively becomes affected by it.

Pathological Anatomy.—Glandular atrophy may be primary or it may represent the terminal period of catarrhal and interstitial gastritis.

Interstitial gastritis may be present in catarrhal gastritis and in proliferating glandular gastritis, and its degree influences the evolution of these forms of gastritis. It may develop before the productive inflammation of the mucous division of the mucosa (catarrhal gastritis), or it may begin during the evolution of catarrhal gastritis. It rarely precedes proliferating glandular gastritis, but is common in the terminal period. It is the only productive process which occurs in gastritis glandularis atrophicans. But in some cases the interstitial inflammation dominates the pathological changes, and

destroys the glands by compression. The changes in the secreting cells are partly degenerative and partly irritative and productive. It is the interstitial inflammation which causes change of form during the evolution of gastritis, and produces atypical clinical cases. If the interstitial inflammation is primary and dominates the evolution of the morbid process, during the period of advanced compression-atrophy, the mucous membrane becomes grayish, smooth, and hard. Here and there, imbedded in the newly-formed connective tissue, may be seen the remains of the gastric tubules. The compression-atrophy is most marked in the pyloric region. The submucosa is thickened and infiltrated, and the infiltration often extends to the muscular layer. The wall of the

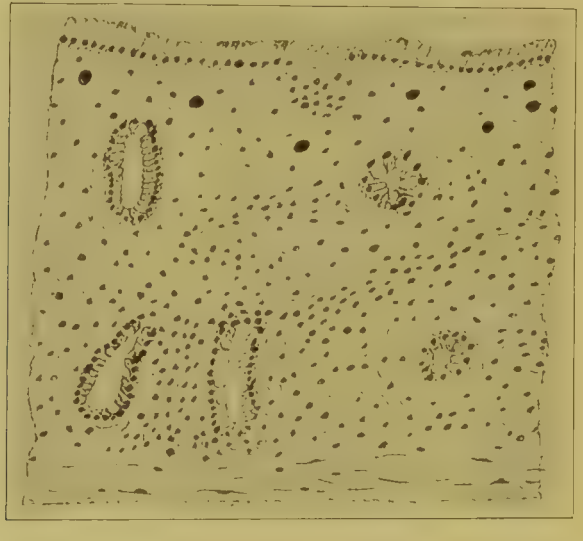


Fig. 23.—Gastritis chronica catarrhalis (terminal atrophy). × 115. (Authors' specimen.)

stomach is hard, thickened, and the stomach is reduced notably in size. Catarrhal gastritis, in the manner already described, may end in complete destruction of the glandular layer.

Glandular atrophy also occurs as the expression of a primary cellular degeneration. The stomach is then usually small and its wall is very thin. The inner surface is smooth and of a pale yellow or reddish color, or sometimes waxy. Gastritis glandularis atrophicans is characterized by degeneration of both the cylindrical cells and the cells which line the glands. Consequently, both the mucous and the glandular divisions of the mucosa are affected by the cellular degeneration. The cylindrical cells are converted into goblet cells

and into columnar cells with striated ends. Some are short and indistinct in outline. The protoplasm loses its affinities for stains, and falls into a pale granular mass. The chief and the border cells can not be distinguished, or only here and there can be seen a cell which preserves its identity. The cells lose their form, diminish in size, yield to the pressure due to the accumulation of degenerate cells, and their cytoplasm loses its affinities for stains and falls into a pale granular mass. The nuclei also undergo chromatosic degeneration. Finally, the mucosa is converted into a soft mass of embryonic cells, mixed with granular masses and hyaline

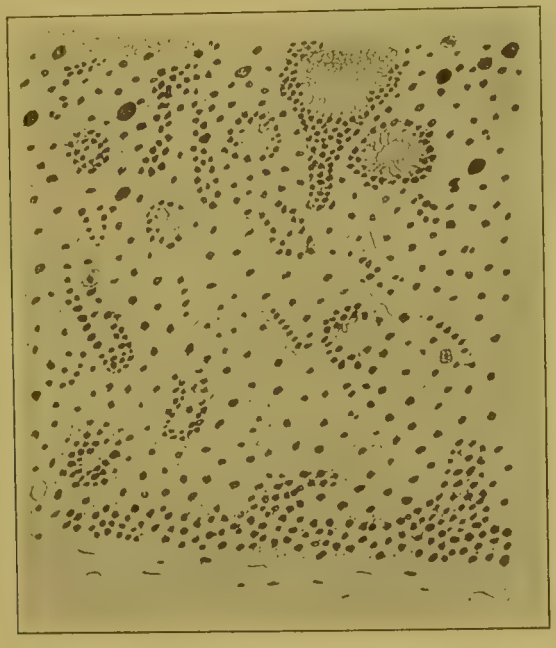


Fig. 24.—Gastritis glandularis atrophicans. × 115. (Authors' specimen.)

bodies, which represent degenerated secreting cells and degenerated wandering leukocytes. Here and there may be seen fragments of partly preserved glands, and in other parts of the mucosa may be found glands whose cells are undergoing degeneration. The surface of the mucosa is without folds, vulnerable, without prominences or depressions, and covered with blood-stained mucus and coagulated protoplasm. The interglandular and subglandular tissues are infiltrated with embryonic cells, with disintegrating leukocytes, with esinophile cells, and with irregularly outlined, flattened, or elongated nucleated cells with granular protoplasm. The submucosa is somewhat thickened, and the walls of the arterioles

in some cases undergo amyloid degeneration. The muscular layer is commonly left intact in the earlier stages, but it may become infiltrated and lose its power. The morbid process is essentially a cellular degeneration without reparative effort, which is accompanied by interstitial inflammation. The final result is complete glandular destruction. Such is atrophy of the gastric glands—a primary and distinct form of gastritis.

Clinical Description.—The evolution of glandular atrophy may be divided into three periods of variable duration: the periods of compensation, of gastric symptoms, and of inanition. The patient is usually in the last third of the normal course of life, but gastric atrophy may occur at an early age.

The period of compensation may be short or may extend over a number of years. During this period there are no digestive symptoms, and the body remains well nourished. The intestines do perfectly the chemical work of the stomach, the integrity of the motor function of the stomach being preserved. The secretion of the stomach is diminished, or even completely lost. The hydrochloric acid and the ferments are all about equally decreased or lost. The quantity of mucus in the test-meal contents and in the lavage water may be increased or it may be diminished. Nutrition is maintained by the intestines without digestive disturbance.

The period of gastric symptoms is also variable in duration, and may exist from the beginning of the disease. These symptoms are often ill-defined, but sometimes characteristic. There may be a little heaviness, or fullness, or flatulency after meals, attended by discomfort, occasionally by bilious or alimentary vomiting, sometimes by diarrhea, but more frequently by constipation. In cases of glandular atrophy, diarrhea is easily produced by dietetic errors and excesses. The vomit sometimes contains blood, particularly when grave oligocythemia is present. The appetite is diminished, often lost, a disgust for meats may exist, but the cereals, fish, milk, vegetables, and fruits are eaten more readily, without, however, relieving materially the distress. During short intervals of two or three weeks the symptoms may disappear, but again return. More characteristic is the digestive pain, which sometimes continues until the stomach is empty, and often begins as soon as the food, or even water, enters the stomach. The pain is almost invariably excited when the stomach is distended with food or by inflation. The pain is sometimes very severe, and when associated with the functional signs of atrophy is of very great diagnostic value. During the period of gastric repose paroxysmal peristaltic pains may develop in the colon. The

epigastrium may be very sensitive. Vomiting is an occasional symptom, and the stomach may become intolerant.

The gastric and the inanition periods are not clearly separated. The two may begin together, or the inanition may commence with intestinal disturbance, particularly diarrhea, and the gastric symptoms appear later. Sometimes the gastric symptoms may never become prominent, or they may only appear periodically after days of comfort.

The inanition is usually progressive, and, unless due to a temporary disturbance of the intestines or to improper diet, it produces death by starvation in six or eight months. The patient becomes weak, pale, cachectic, and sometimes neurasthenic. Dyspnea and palpitation occur on the least effort. The mind becomes quickly tired, and insomnia is often obstinate. But emaciation may not be great, as the bloodless and inactive patient burns but little of the body fat. Nearer to the fatal termination, the fat in the food is better absorbed by the intestines than is any other food. Inanition delirium and sometimes coma, precede the death of the prostrated, bloodless, cachectic, and starved patient.

Symptomatology.—The loss of appetite, the vague digestive symptoms, vomiting, constipation, and diarrhea are common to a number of severe chronic diseases of the stomach, and have little diagnostic value. Of more importance are the pain and inanition. Severe paroxysms of pain, usually digestive, but rarely occurring when the stomach should be empty, and then, as a rule, excited by a reflux of bile, associated with greatly diminished or lost gastric secretion, and with preservation of the motor function, should arouse suspicion of glandular atrophy. Pain, in association with nearly lost gastric secretion, occurs in carcinoma, but is very rare in chronic asthenic gastritis and adenasthenia gastrica.

The inanition may become very pronounced, be even fatal, and characterizes one of the stages of the disease. Its coexistence with motor sufficiency of the stomach, and with the employment of a diet containing nutriment enough to meet the requirements of the body, and its increase and decrease in relation with the digestive and absorptive work done by the intestines, are distinctive features. It may be progressive in spite of every effort to nourish the patient, and may be accompanied by the usual signs of grave oligocythemia. Emaciation does not occur until intestinal digestion and absorption begin to fail, unless the diet is insufficient. The body fat may be preserved even when the patient is almost bloodless.

The severe anemia is generally secondary. The total

quantity of the blood is diminished, but there are often no hemic murmurs, and the hemoglobin percentage is less reduced, as a rule, than that of the red corpuscles. There is leukopenia, but the polynuclear white cells largely predominate, and there are megalocytes, poikilocytosis, and microcytes, but no megaloblasts. Lymphemia and nucleated red corpuscles of normal (normoblasts) or of abnormal (microblasts, megaloblasts, gigantoblasts) sizes are rarely found in the secondary grave oligocythemia of atrophy of the gastric glands. As a result of the disease of the blood there may be hemorrhages in the retina, from the mucous membranes, and in the structure of the various organs. In a case of the authors, with hemorrhages but no megaloblasts, the liver and kidneys contained no accumulation of iron, and the stomach was small, thin, and the gastric glands were completely atrophied. The glands of the stomach may undergo fatty degeneration in grave oligocythemia due to other causes, just as do the kidneys and the heart muscle. The anemia caused by atrophy of the gastric glands is simply a grave secondary inanition anemia, accompanied by an altered plasma which becomes hematocytolytic, and by diminished resistance and insufficient development of the red and white corpuscles.

The physical signs are of little diagnostic value. The stomach is usually small and does not splash abnormally. The epigastric tenderness may create a false suspicion of ulcer or of carcinoma. The physical signs possess chiefly a negative diagnostic value.

The functional signs of atrophy of the gastric glands constitute, clinically, its chief distinctive features. The motor function is normal, and if the food be fluid and finely divided it usually leaves the stomach even earlier than in health. But motor insufficiency does not necessarily exclude glandular atrophy, as the motor insufficiency may be an accidental association, produced by other causes, but never by the glandular atrophy. There is no delayed evacuation of the stomach produced by excessive secretion, or by continuous secretion, or by obstruction, or by spasm of the pylorus, or by weakness or infiltration of the muscular layer. The stomach grows small as its wall becomes thin.

After the test-breakfast the contents contain neither HCl (free or in organic combination), nor pepsin, nor labferment, nor the mother substances of the ferments. There may be little or no excess of mucus, unless it be swallowed, and only a few c.c. of water or secretion fluid. Such is the almost total absence of secretion when the atrophy is complete.

When incipient or partial, all the products of secretion simply diminish more or less.

In the expressed contents—small in quantity, thick, containing little water, with the roll, as swallowed, unchanged physically, covered with mucus or mixed with a little slimy fluid—are found none of the products of pepsin-hydrochloric digestion. Starch digestion is normal, and the reaction with Fehling's solution is positive.

There are no characteristic bacteriological signs. The germs that are found have been swallowed. There may be a trace of lactic acid (ether extract), but never after previous lavage. Sometimes there is a little butyric acid. The total acidity may vary from zero to 8 or 10. The sweetened contents yield no gas in the fermentation tubes.

Blood is sometimes vomited, and usually comes from congested vessels about the cardia. An important anatomical sign of differential diagnostic value is the absence of well-preserved gastric epithelium in the morning washings from the stomach. The tube is rarely used without obtaining a little bloody exudate, and, frequently, pieces of the degenerated mucous membrane, goblet cells, and leukocytes from the interglandular tissues.

Neither digestive leukocytosis nor the diminished acidity of the urine secreted during the period of functional activity of the stomach occurs. Digestive leukocytosis, however, occurs during the stage of compensation and before inanition and anemia develop.

Differential Diagnosis.—Atrophy of the gastric glands may be confounded with adenasthenia gastrica, chronic asthenic gastritis, and carcinoma.

Adenasthenia gastrica is most common in hysterical or neurasthenic women, between the ages of fifteen and thirty-five. Gastric atrophy is more common in men after the thirty-fifth year. The dynamic affection is often remittent in its course, being accompanied by rapid changes in secretion. Atrophy of the gastric glands, after digestive compensation is lost, often runs a rapid and progessive course to a fatal termination within a year. Secretion once lost is never recovered. In adenasthenia gastrica the acid secretion varies from a slight decrease of the normal quantity to a total temporary loss. There may be little or no free HCl in the contents after the test-breakfast, or only combined hydrochloric acid, or none whatever. The secretory activity of the stomach now drops, now rises rapidly, and may nearly always be excited so as to give plain ferment tests. The secretion of mucus in

adenasthenia gastrica is never increased, and may not be even altered when the acid secretion is completely suppressed. The morning washings of the stomach contain well-preserved and perfectly staining gastric epithelium, which is never found in glandular atrophy. In the dynamic affections there is no vomiting, no paroxysmal pain, or, if the diet is sufficient, there is no emaciation, inanition, grave anemia, nor cachexia, nor signs of an anatomical disease of the mucous membrane. Indeed, the two diseases are not likely to be confounded, except during the compensation stage of glandular atrophy. Moreover, adenasthenia gastrica never produces persistent achylia. The disproportion between the glandular destruction in cases of so-called "simple achylia" and the complete suppression of secretion is no proof of the nervous nature of "simple achylia." There may be complete suppression of secretion in severe asthenic gastritis accompanied by acute or subacute interstitial inflammation. Interstitial gastritis may reduce the excessively rich secretion of hypersthenic gastritis to hypochylia. In both cases the chief and border cells may be in perfect preservation, and this condition is, consequently, out of harmony with the depression of secretion. The nervous system may be highly toned without influencing secretion in so-called nervous or "simple achylia." Where pieces of mucous membrane, in our experience, have shown the partial preservation of the peptic glands and cells, there has been marked interstitial infiltration, and, usually, advanced changes in the mucous division of the mucosa; or other pieces obtained from the same cases have shown the characteristics of glandular atrophy. We can not admit, therefore, the existence of persistent achylia as a dynamic affection. Secretion may sometimes be restored by treatment when the achylia is due to interstitial gastritis.

Some cases of carcinoma of the stomach may be readily mistaken for glandular atrophy. Cancer nearly always produces motor insufficiency and an active bacillary germ growth. This is a valuable differential sign, and is conclusive when associated with lactic acid formation. But motor insufficiency may not develop until late in the evolution of some cases of cancer, and may never develop in some cases of scirrhus. The ferments do not completely disappear until late in the cachectic stage of cancer. The blood changes are unlike those of gastric atrophy. Hemorrhage is more frequent, pain is more constant, and a tumor can often be detected. Mucus in large quantity, and cancer cells or exfoliations, may be found in the vomit, in the morning washings, and in the ex-

pressed contents after a test-breakfast. The clinical history, particularly the state of the health of the patient when the trouble began, may often give weight to the one or the other side. Both diseases may be progressive and may rapidly terminate in death by inanition, the inanition being toxic (protoplasm poison) in the one and intestinal in the other. The differentiation must be made by a careful study of all the circumstances, symptoms, and signs. Some are found only in the one or the other disease; some are more common in the one than in the other. The ill-defined atypical cases of the two diseases may so closely resemble each other as to leave the physician in ignorance and doubt. Atrophy may be a complication of cancer, developing during the cachectic period.

Chronic asthenic gastritis is a painless disease; the digestive symptoms vary with the quality (solid or liquid) of the food, and mucus is secreted in excess. In the uncomplicated cases the nutrition of the body is well preserved, and there is properly no extreme emaciation nor grave, progressive anemia. The course is long and tedious. These symptoms and signs are all in contrast with those of glandular atrophy, which may, however, develop as the anatomical termination of chronic asthenic gastritis. The clinical history and evolution are then the chief distinctive features. Both these forms of atrophy may be accompanied by paroxysms of pain. Chronic hypersthenic gastritis is widely different in its manifestations, evolution, and objective signs from atrophy of the gastric glands.

Prognosis.—Atrophy of the gastric glands may not in itself seriously compromise the general health nor shorten life. It calls into activity all the reserve forces of digestion, but the healthy intestines stand between it and inanition. With good digestive hygiene and a proper diet, digestive compensation need not be disturbed. But even when completely compensated, the disease constitutes a weakness, and opens the portals to gastro-intestinal infection.

When digestive compensation is destroyed and the intestine fails to do its work, the situation becomes grave. The prognosis is bad when the inanition and anemia are marked and are uncontrollable by proper treatment. The disease is in the stomach, but the prognosis is given by the intestines, by the state of nutrition and of the blood, and by the complications, and, when the glandular atrophy is secondary, by the causative disease. Atrophy of the gastric glands does not render the preservation of good health impossible, but it

is a constant menace, and may lead to death by inanition within a few months.

Treatment.—In the treatment of atrophy of the gastric glands physical remedies have no place. Lavage, the intragastric douche, intragastric electrization, and massage are not only without purpose, but may injure the exceedingly vulnerable mucous membrane. The stomach must be protected at all hazards, for acute inflammation may be excited and the motor function thus impaired. No effort need be made to excite or to restore secretion. This function of the stomach is irreparably lost. Physiological treatment is useless and harmful. There is no hope of making a degenerate cell work, or of restoring its waning vitality by stimulation and excitation. Excitant medication only hastens the death of the cell, or if the cell be already destroyed excitation is altogether out of place. The only local treatment of a degenerate cell which is beneficial is most scrupulous and vigorous protection. But the motor function of the stomach should be carefully guarded and strengthened.

This can best be done by careful digestive hygiene, hydrotherapy, faradism, and in some cases by the use of a few drugs. Rest before and after each meal, the avoidance of overloading the stomach, and the exclusion of innutritious, indigestible, and irritating food and drinks are always necessary. Water should be used to tone the neuromuscular systems. The Scottish douche over the abdomen and lower extremities is an excellent procedure the value of which is proven by clinical experience. External faradism may also be employed. The object is twofold—to diminish the work required of the gastric muscle and to keep the muscle more than equal to its work.

The next object is to aid and to protect the intestines, thus securing and maintaining digestive compensation. The abdomen should be carefully guarded by proper clothing against extremes and changes of external temperature. The food should be finely divided, digestible, and utilizable, freshly prepared and sterilized; no coarse, irritating food or drinks, and no food liable to ferment or to putrefy. The same policy of careful protection of the intestines should extend to drugs —no irritants, nothing to disorder digestion, no purgatives, and particularly no salines. The bowels should be regulated by the diet, intestinal massage, electricity, and the employment of small laxative rectal injections.

The work of the intestines may sometimes be lightened by the employment of pancreatin and a little soda or a fresh ex-

tract of pancreas. Papoid sometimes helps. The administration of hydrochloric acid and pepsin, contrary to what might theoretically be expected, does no good. Chemical treatment would seem to be strongly indicated, and theoretically this is so. Give pepsin and hydrochloric acid, and give them in sufficient quantities to replace the stomach's lost digestive power, is the plausible rule of practice. Our experience with this method forces us to oppose it obstinately. In some cases it does no very obvious harm, but it does not relieve the subjective symptoms, if they exist; it sometimes excites discomfort; it does not improve nutrition; but it often disturbs intestinal digestion; it often increases intestinal putrefaction and, consequently, auto-intoxication; it arrests ptyalin digestion in the stomach; and it irritates the vulnerable mucous membrane. If chemical treatment is to be employed it is better to give pancreatin and a little soda, a fresh extract of pancreas, or a vegetable ferment.

The patient should be as well nourished as possible, and under no circumstances should an insufficient diet be prescribed. The disease is in the stomach, but a danger is in starvation.

The diet is selected by the stomach, by the needs of nutrition, and chiefly by the intestines. It is the intestines which, in this disease, have all the food to digest and utilize. The food should be easily evacuated by the stomach, digestible and utilizable by the intestines, and sufficient to supply the demands of the body for nutriment.

The food should be all very finely divided, fresh, and nutritious. Sterilized milk, if well borne, and the fermented preparations of milk are suitable. The cereals—rice and the preparations of wheat and of Indian corn are the best—should be cooked all to pieces. Only bread thinly sliced and browned through and through should be permitted. Expressed meat juice, meat broths (small quantity), or clear vegetable soup (either may be thickened with a thoroughly cooked cereal), meat powder, and lean, short-fibered fish are well borne. The lean pulp (Enterprise chopper, old pattern) of the fresh meats and fowl and game—beef, mutton, chicken, white meat of turkey, quail, etc.—may be made into cakes and broiled with a little butter. Eggs often disagree, particularly when much of the yolk is eaten. Spinach, string beans, and fresh garden peas should be thoroughly cooked and passed through the colander or sieve. The juice of a ripe orange, or of a few grapes, or a baked apple may sometimes be permitted. Fresh butter, and cream, and breakfast

bacon are the best forms of fat. Cod-liver oil is suitable if it agrees. Coffee, very weak tea, "vigor chocolate," sometimes cocoa, and plain water usually also agree very well. From this list a mixed and supporting, or even restorative, diet may be selected.

When inanition or anemia begins rest should be ordered to diminish the requirements of nutrition, and the diet should be supplemented by rectal feeding. Every effort must be made to maintain the volume and richness of the blood, and an intestinal complication should receive prompt treatment.

Briefly stated, the chief indications to be met in the management of chronic atrophy of the gastric glands are: (1) Protect the stomach against all mechanical and chemical irritation; (2) preserve and strengthen and favor its motor function; (3) maintain intestinal digestive compensation; (4) maintain the nutrition of the body, the composition of the blood, and the tone and strength of the neuromuscular systems.

CHAPTER II.

ULCER OF THE STOMACH.

Ulceration of the gastric mucosa may occur as an episode in the evolution of acute and chronic gastritis and during the course of neoplasms of the stomach. The mucous membrane may be studded with punctate erosions; or there may be small ulcers of the size of the head of a pin which extend into or through the mucosa; or there may be superficial and, rarely, deep ulcers running along the course of obliterated blood-vessels. The formation of the erosions of gastritis may often be revealed by the little exfoliations or sloughs, which are rarely larger than $\frac{1}{5}$ of an inch in diameter, and which are most readily found in the early morning lavage water. The neoplasms of the stomach may also ulcerate. Insignificant or profuse hemorrhages may accompany these forms of secondary ulceration. Primary ulceration may occur as a local bacterial infection in anthrax, in tuberculosis, and in syphilis. Tubercular ulceration of the stomach is rare, and is accompanied, as a rule, by pulmonary or intestinal tuberculosis. We have been able to collect only 23 authentic cases. Syphilitic ulcer of the stomach is also rare; and, like the

tubercular ulcer, it is a postmortem discovery, and is consequently of no great interest to the physician. Such is not, however, the case with simple ulcer of the stomach.

Ulcer of the stomach is an anatomical disease, characterized by a sharply limited defect of the mucous membrane, distinct in its genesis, its form, and its evolution. The disease has been given many other names descriptive of some striking peculiarity or embodying a theory of its genesis—simple, round, chronic, perforating, corrosive, rodent, hemorrhagic, peptic.

The most frequent functional sign is digestive hydrochloric superacidity, but this sign is neither constant nor characteristic. As a clinical entity it is a modern disease, and is characterized in its typical form by gastric pain, by hemorrhage, and by vomiting. These cardinal symptoms are not constant, and are characteristic only when possessing a number of distinctive features. Beneath the play of expression the unity of the disease is preserved by the gross anatomical and microscopic characters of the localized destruction of the mucous membrane, tending to go deeper and to involve the other layers.

Frequency.—The frequency of gastric ulcer varies in different parts of the world. Most English and American authors state that in about 5 per cent. of deaths from all causes an ulcer or an ulcer scar can be found. According to Lebert, in about 4 per cent. of the autopsies made in middle Europe either an open ulcer or a cicatrix is found. Of 41,688 cases treated in the clinics of Zurich and of Breslau by Lebert, about 0.6 per cent. had gastric ulcer. In hospital practice McCall Anderson met with 35 cases of ulcer in 2538 medical cases, or about 1 in 73. The statistics of autopsies collected by others give important variations from these percentages—for instance, Dahlaup (Copenhagen), 13 per cent.; Nolte (Munich), 1.23 per cent.; Waldeyer (Breslau), 1.6 per cent.; Lebert (Breslau), 2.5 per cent.; Jaksch (Prag), 3.2 per cent.; Jaworski and Korczynski (Krakow), 0.24 per cent.; Hauser (Erlangen), 3.4 per cent.; Steiner (Berlin), 3.6 per cent.; Berthold (Berlin), 2.7 per cent.; Starke (Jena), 10 per cent. Age, sex, occupation, diet, and the prevalence of gastric troubles associated with excessive secretion may explain the differences of the statistics. The personal equation of the pathologist doubtless also comes into play, some searching with greater interest and care for small ulcer scars. Cases are likely to be included in the statistics where the stomach was not carefully examined. In a large private practice, con-

DEATHS FROM ULCER OF STOMACH IN NEW YORK CITY FOR TEN YEARS, BY AGE AND SEX.

(Official Tabulation.)

Year.	Sex.	Under One Year.	One and Under Two.	Two and Under Three.	Three and Under Four.	Four and Under Five.	Five and Under Ten.	Ten and Under Fifteen.	Fifteen and Under Twenty.	Twenty and Under Twenty-five.	Twenty-five and Under Thirty-five.	Thirty-five and Under Forty-five.	Forty-five and Under Fifty-five.	Fifty-five and Under Sixty-five.	Sixty-five and Under Seventy-five.	Seventy-five and Under Eighty-five.	Eighty-five and Over.	Total.
1887	M.								1		4	6	6	4	2	1		24
	F.							1		4	5	3	3	3	2			21
1888	M.								4		7		12			4		27
	F.								4		6		9			1		20
1889	M.	1							3		18		13			3		38
	F.		2						4		18		7			2		33
1890	M.	1				1	1		1	1	3	3	6	8	6	1		29
	F.								2	2	3	2	8	1	4	1		26
1891	M.	1		1					1		1	2	9	1	3	1		20
	F.	1					1		3	4	8	6	3	1	2			29
1892	M.						1	1	1	1	7	9	7	4	2	1		34
	F.	2						1	3	3	8	4	8	5	5	1		40
1893	M.										6	9	6	4	1			26
	F.								2	6	2	5	8	5	4	3		35
1894	M.				1				1	1	4	7	8	6	1	3		32
	F.				1				1	4	6	7	6	3	3	1		32
1895	M.						1		1	3	8	11	12	3	2	2		43
	F.								1	3	7	4	3	8	2			28
1896	M.						1			1	6	10	9	4	1	1		33
	F.	1					1		4	5	10	6	8	8	4	1		48
		7	2	1	2	1	6	3	22	38	88	94	110	68	44	27	0	618
									15		49		41					

sisting almost exclusively of patients with gastric and intestinal diseases, we have found simple ulcer in about 1 in 134 cases.

Of the 618 deaths from ulcer in New York City (see Table) during the decad 1887 to 1896, 306 were male and 312 were female. As regards the age at which death occurred, 97 were under twenty-five years; 231 were between twenty-five and forty-five years; 219 were between forty-five and sixty-five years (or 450 between twenty-five and sixty-five years); and 71 were between sixty-five and eighty-five years.

Average population of N. Y. City for decad 1887–1896.	Average number of deaths annually from all causes, 1887–1896.	Average number of deaths annually, from ulcer, 1887–1896.
1,781,301	41,738	62

Population N. Y. City, 1896.	Total deaths, N. Y. City, 1896.	Deaths from ulcer, N. Y. City, 1896.
1,934,972	40,557	81

According to these statistics, 1 in every 763 of the total deaths (1887–1896), or 1 in every 500 of the total deaths (1896), is due to ulcer of the stomach; or 1 in every 28,730 living inhabitants (1887–1896), or 1 in every 23,888 living inhabitants (1896). The death-rate of ulcer, like that of cancer (Bryant), is increasing from year to year. The increase is not due to accident, or to worse methods of treatment, or to the greater and greater frequency of ulcer; but the increasing death-rate may, in all probability, be explained by greater accuracy of death reports.

Etiology.—The occurrence of gastric ulcer is in close relation with the age, some periods of life being remarkably free from the disease and others conspicuous on account of its frequency. Certainly, these variations can not be attributed to the influence of age itself, but are due to the prevalence at certain periods of life of the many predisposing causes more closely connected with the genesis of the trouble.

Simple gastric ulcer is very rare before the tenth year. This exemption may be due in part to the diet, to the rarity of chronic disease of the stomach accompanied by the secretion of an excessively active and acid gastric juice, to healthy arteries, and to the great resistant and reparative powers of the young. It is also supposed by some that an ulcer, should it occur, would likely remain latent, and heal rapidly under the influence of the bland diet employed in the digestive disorders of infancy and childhood. But autopsies made on children before the tenth year reveal, with comparative rareness, either an ulcer or an ulcer scar.

In drawing conclusions from statistics showing the relation of age to the genesis of simple ulcer of the stomach, errors are likely to be made. Evidently, the cases where only cicatrices are found must be deducted from the statistics of autopsies, for the time when the ulcer or its scar is found does not represent the date of its commencement. One of the clinical characteristics of ulcer is its chronicity, and the ulcer or its scar may be unexpectedly discovered postmortem a long period after its beginning. If death be due to the ulcer, and the clinical history be known, still, the early period may have been entirely latent. After the correction of the statistics it will be found that more than one-half of the cases begin between twenty and thirty, and about four-fifths between twenty and forty. About nine-tenths of the deaths from gastric ulcer occur between twenty and sixty, but the period of greatest mortality is between forty and sixty. After the sixtieth year the frequency and the mortality are low, as few cases which began at the age when the disease predominates are brought over and credited to the later period. Ulcer in old age is a very fatal disease, on account of the low healing power when life's course is nearly run; but it is not a frequent disease, possibly on account of the low hydrochloric acidity of the gastric juice.

Simple ulcer of the stomach is more frequent in the female than in the male, but the statistics taken in different localities at different times give inconstant results. This is very apparent in the statistics of Lebert. Of the 41,688 patients treated in the clinics, 21,020 were men (69 cases) and 20,688 women (183 cases). According to these figures, the disease is nearly three times more frequent in women (0.32 per cent. to 0.88 per cent.). In Breslau the proportion is 1 to 3.5, but in Zurich it is 3 to 4, in favor of its predominance in women. McCall Anderson, in his statistics of 2538 medical cases, of which 927 were women and 1611 men, records that 32 of the 35 cases of ulcer occurred in women (1 in 29) and 3 in men (1 in 537). Most authors, including Brinton, give the proportion as about 1 to 2, but Steiner says 8 to 11, and Willigk proposes 1 to 3 in favor of its greater frequency in women. After the fiftieth year ulcer is more frequent in men than in women. Many circumstances may explain the differences in the statistical reports; such as age, occupation, constitution, diet, and the predominance in women of certain diseases which act as predisposing causes of ulcer, such as gastroptosis, hysteria, and chlorosis. Ulcer is more frequent in hysteria and less frequent in chlorosis (one per cent.,

Hayem) than is popularly believed. Rasmussen claims that tight lacing produces a furrow across the stomach beneath the edge of the right costal border, and he states that ulcer develops frequently along this furrow. Some predisposing diseases are more frequent in men—*e. g.*, arteriosclerosis and hypersthenic gastritis. It is difficult to estimate the exact influence of sex, but clinical and postmortem observation establishes the greater frequency of the disease in women.

Occupation and the social and financial condition seem, directly or indirectly, to exert an influence in the causation of ulcer. It is well known that the disease is frequent in cooks, supposably on account of the pernicious habit of tasting hot dishes, of irregular eating, and, possibly, on account of reflex secretion, and of the presence in the stomach of a quantity of uncombined acid. A delayed dinner in this way often produces a gastric headache. Cooks are often affected with adenohypersthenia, a favorable condition for the development of ulcer in conjunction with a local disturbance of circulation and of nutrition. Traumatism, associated or connected with the occupation, is a causative influence, much more frequently active than is commonly believed. House girls are more subject to gastric ulcer than is any other class, since they are so often anemic or chlorotic, and suffer from menstrual disorders. The disease is much more frequent among household servants than among the rich, with the exception of the clinical form of ulcer developing in the anemic girl and proving rapidly fatal by perforation. It is rare among those who live and work in the open air, and who eat largely a vegetable diet. The disease is almost unknown in parts of Russia, in the Rhone Valley, and among the Bavarian Alps, inhabited by a poor peasantry who rarely get an opportunity to taste meats or highly-sweetened articles of food. Yet the disease is very rare among the negro field-laborers of the South, who live on cornbread, molasses, and bacon. The character of the diet is without influence in the production of the disease, unless, in combination with other factors, it engenders chronic hypersthenic gastritis. Alcoholism and iced drinks have not the etiological influence often attributed to them. Ulcer respects no class, nor condition, nor vocation, but develops most frequently in those who are predisposed to local disturbances of the circulation or to a local trophic defect; and it complicates most often the diseases, local or general, which are accompanied by a richly acid gastric secretion.

Age, sex, occupation, diet, and the social and financial con-

dition are only predisposing causes. More directly and closely related to the genesis of the ulcer are certain cardiovascular diseases, diseases of the blood, infectious diseases, and diseases of the stomach.

The influence of diseases of the heart and the blood-vessels is unquestionable, ulcer being often the consequence of arteriosclerosis, embolism, thrombosis, hemorrhagic infarct, and intense local inflammation. The circulation of a circumscribed area is thus cut off, resulting in local death, and in the digestion of the dead tissue. It has long been claimed, both on clinical and anatomical grounds, that chronic ulcer may be caused by a local disease of a blood-vessel of the stomach. This may occur in arteriosclerosis, in the diseases of the red corpuscles, in syphilis, and such other diseases as produce inflammatory or degenerative changes of the arteries. As a result of the fatty degeneration of the arterial wall, of a small aneurysm, of arteriosclerosis, or of endarteritis, a small arterial branch becomes plugged or obliterated, and the area of the mucosa fed by this small artery dies and is cast off or digested, leaving an ulcer of the peculiar shape and form of chronic ulcer of the stomach. This explanation of the genesis of ulcer has been ably defended by Virchow and others. It is denounced by some pathologists on the grounds that ulcer is rare in old age, when arterial disease is frequent; that it is frequent before the age of forty, when disease of the arteries (non-syphilitic) is rare; that ulcer occurs frequently when there is no disease of the arteries; that diseases of the arteries exist so frequently without an ulcer forming throughout their long course; that arteriosclerosis may produce atrophic gastritis without causing an ulcer. These facts should be given their due weight; but it is not claimed that all ulcers are produced by arterial disease, and it is known that some gastric ulcers are caused in this manner. The obliteration of a small artery, be it due to disease of its walls, to embolism, to thrombosis, or to a node-like and intense inflammatory infiltration and compression, may be the cause of ulcer, just as a hemorrhagic infarct may produce the death of a circumscribed part of the mucosa which lies over it. Ulcer is not a disease with a single cause and one mode of genesis, as is often assumed.

The etiological influence of the diseases of the blood is questioned by many authors, who consider the blood changes secondary. It is doubtless true, if the evidence of close clinical study is trustworthy, that the anemia is often hemorrhagic or due to inanition, the ulcer being primary, but prevented

from healing by the dystrophic influence of the thin blood. This is well shown by the evolution of the anemia and by its rapid cure by controlling the hemorrhage and the inanition. That a supposed primary anemia may be the consequence of a latent ulcer is also a possibility. It is equally certain that the anemia is often primary and causative, or is a coeffect. The existence of chlorosis can be explained only as a coincidence or as a cause, for neither hemorrhage nor inanition will produce it, and ulcer is far more frequent in chlorosis than in oligocythemia. The diseases of the blood are supposed to act by producing a local arterial spasm or fatty degeneration of the arterial walls. Hydrochloric superacidity is not rare in chlorosis, and could at least make the genesis of the ulcer easier. It may be taken as a clinical fact that oligocythemia and chlorosis may aid in the generation and often play a predominant part in the persistence of ulcer.

It has been maintained by some that ulcer is a specific process, and bacteriology has been called upon to reveal the unique cause. That ulcer may result from an infectious disease seems established by clinical observation and by experimental pathology. Cases have been reported following diseases associated in their evolution withpus formation or the production of hemorrhagic infarcts or thrombi, and the pathogenic bacteria have been found in the blood-vessel where the necrotic process was localized (Letulle). Puerperal fever, typhoid fever, endocarditis, abscess, suppurative peritonitis, pleurisy, tuberculosis, and syphilis, among other infectious diseases, have been mentioned in this connection. It may be admitted that gastric ulcer may be produced by infection through the circulation. The presence of bacteria in the walls of the ulcer (Boettcher) can only be considered accidental and secondary.

The pathogenic influence of hydrochloric superacidity is still a matter of dispute. Some pathologists make this condition essential, and claim that without it ulcer does not occur, or, at least, that no cases which contradict this hypothesis have yet been reported which do not admit of explanation by the well-known intermittent course of the diseases or the affections of the stomach accompanied by excessive secretion. The theory is seductive in its simplicity—a very active and superacid secretion, a diminution of the vitality of a localized area of the mucous membrane, autodigestion and a resulting circumscribed defect of the mucous membrane, the repair of which is delayed or prevented by the irritation or the action of the strongly acid gastric juice. But if its parts be more

closely examined, it would seem that the theory is too exclusive.

There is no evidence to prove that excessive hydrochloric acidity always precedes the development of ulcer. Simple adenohypersthenia (a dynamic affection) is rarely complicated by an ulcer; it is, however, different, as we have seen, with hypersthenic gastritis. It is a fact of which we have no doubt that ulcer may run its entire course without excessive hydrochloric acidity, and where the excessive secretion is not suppressed by hemorrhage, weakness, inanition, or anemia. When in the course of treatment these symptoms disappear, the excessive secretion does not return. The hyperchlorhydria often disappears permanently with the cure of the ulcer, suggesting at least the possibility of its having been excited in a manner probably analogous to the excessive lachrymal secretion in a corneal ulcer, or that its cause (ulcer, or not) was removed by the ulcer treatment. That it is not alone an efficient cause is proven by the whole mucous membrane not being involved, and by cases of hyperchlorhydria running a long course without ulcer being generated, even after injury of the mucous membrane by the use of the tube. In about 70 per cent. of the cases of ulcer there is hydrochloric superacidity. In the remainder of the cases secretion is normal, or there may be hypochylia, or, very rarely, even achylia. The hydrochloric superacidity may have preceded the ulcer; it may have followed the ulcer (obstruction of pylorus, etc.); or it may be the irritative expression of the ulcer. It would not be right to assume that at the moment when the ulcer developed there was not, in any of a series of cases, hyperchlorhydria, but it is not highly probable that a temporary hyperchlorhydria would produce an ulcer. Moreover, ulcer is not situated most frequently where the gastric secretion is most acid nor most in contact with the mucous membrane; but where all anatomical alterations of the mucous membrane are most frequent and most intense. Nor can it be admitted that the occurrence of this peculiar form of ulcer only in the stomach and in the digestive tube close to it, is proof that the gastric juice has anything to do with its causation. Certainly, peptic ulcer can not be produced by autodigestion alone.

Autodigestion is prevented, not by mucus, not by the alkaline blood nor by active absorption, but by the resistance of the living protoplasm of the cell. The mucus does not prevent postmortem digestion nor the peptonization of meat coated by it. The gastric juice is secreted beneath

the mucus, and does not digest a part of the mucous membrane which is kept free from mucus. Erosions heal and cells are regenerated in the presence of the gastric juice. Germs live in it. Ferment-producing organisms do not digest themselves, and the blood and the tissues of the body destroy ferments. The action is not prevented by the alkaline blood, for the HCl secreted by and in the cell is not neutralized; the pancreatic juice does not digest the intestines, nor does papain digest either an acid or an alkaline-reacting living tissue. Autodigestion is possible only where the protoplasm of the cell no longer possesses its normal properties and powers of resistance.

Though excessive hydrochloric acidity is not a necessary factor in the genesis of ulcer, it can not be doubted that it is often a pathogenic power of great importance, and often exerts a pernicious influence on the persistence of the trouble; and its control is a commanding indication in the treatment. Where the excessive secretion is a symptom of hypersthenic gastritis, the condition exists most favorable to the genesis of ulcer; the node-like and limited infiltration of the mucous membrane or submucosa being often sufficient to reduce the circulation and nutrition of a local area of the mucous membrane so low as to render it unable to resist the digestive power of the excessively acid and very active gastric juice.

Etiologically, ulcer is primarily dystrophic, the essential conditions of its genesis being the local defect of the circulation and of the nutrition in a part exposed to the digestive action of a gastric juice usually excessively acid. The defect is made persistent by the diminished reparative powers of the organism, and by local irritations intimately associated with the functions of the stomach. The etiology gives the commanding indications in treatment—functional rest, protection, and the improvement of nutrition.

Pathological Anatomy.—Simple ulcer of the stomach is usually single, but in about one-fifth of the autopsies more than one is found. These may be of different ages, or a fresh ulcer may be found coexisting with an old scar. Sometimes two old ulcers are found corresponding with the areas supplied by two branches of a small artery (obstructed), which later unite to form one irregularly shaped ulcer. Consequently, a single ulcer or a single scar found at the autopsy may represent the union of two ulcers distinct in their earlier stages. Two, three, four, five, or even more may coexist, and may vary in form, in size, and in age. But cases of multiple peptic ulcers exhibiting these characteristics are rare.

Ulcer shows a decided preference for certain regions of the stomach. The favorite localities are the smaller curvature, the posterior wall, and the pyloric region. The portions most frequently affected are a small area of the posterior surface near the pylorus, and another along the lesser curvature. Often one is located on the lesser curvature, and another close by on the posterior wall. Rarest in the fundus, it is much less frequent on the anterior wall, in the cardiac region, and along the greater curvature than over the favorite localities. About half the cases occur in the pyloric third of the stomach. A plane passing vertically through the cardiac orifice and the tip of the cartilage of the left tenth rib would locate about four-fifths of the ulcers to its right. Brinton gives the following percentages: Posterior surface, 43 per cent.; lesser curvature, 27 per cent.; pyloric extremity, 16 per cent.; anterior and posterior surfaces, 6 per cent.; anterior surface, 5 per cent.; greater curvature, 2 per cent. Welch locates more on the lesser curvature (36 per cent.) than on the posterior wall (30 per cent.), and Lebert gives nearly the same percentage (33 per cent.) for the lesser curvature as does Welch.

The typical peptic ulcer is round or oval, but this form is by no means constant. The borders commonly run in regular curves, but the coalescence of adjacent ulcers may produce a variety of shapes. A comma shape is not rare, and the pylorus may be partly or completely surrounded by a ring. A marked characteristic is the arrangement of the long axis of the ulcer in the direction of the obliterated artery, the area of the distribution of which corresponds with the form and the extension of the ulcer. The size is likewise very variable, the common size being that of a silver dime or quarter; but some are as small as peas, and, exceptionally, the defect may cover a space as large as the adult hand.

The other gross anatomical characteristics present slight variations, dependent, seemingly, on the age, the depth, and the genesis of the ulcer. The borders marked by the mucous membrane are usually perpendicular or slightly rounded, and sometimes a little undermined; but typically the appearance is that of a defect left by the removal of a piece of the mucous membrane with a round chisel gouge. The border is usually uninflamed, but may be red and swollen, and is sometimes hard and calloused and formed of new connective tissue. The ulcer may extend no deeper than the mucous membrane, the bottom being smooth and non-granulated. But often the process begins or extends

deeper, and a remarkable appearance results, the ulcer extending obliquely in a funnel shape, one side of which, particularly, may form a stairway descending to the peritoneum, the steps being formed by the mucous membrane, the submucosa, and the muscular layer. But often the descent is gradual, oblique, conical, with here and there little projections of connective tissue. The bottom is grayish-yellow, or is composed of the pale tissue of the layer to which the ulcer extends; or, it may be occupied by a grayish-black slough. In complicated cases the tissues of an adjacent organ, particularly the pancreas, may be seen in the bottom, or near the apex may be visible the gaping blood-vessel which has caused the fatal hemorrhage.

The gross anatomical characteristics of the recent ulcer would suggest a defect produced by the digestion of the dead tissue corresponding with the distribution of a small arterial branch, without being followed immediately by inflammatory reaction or by an effort at repair. But sooner or later the edges, the wall, and the base are the seat of a productive inflammation. The early stage of the ulcer reveals its dystrophic nature. The inflammation is seldom intense, but, exceptionally, the tissues near the ulcer are secondarily invaded by bacteria. Microscopically, the inflammation about the ulcer is productive, and presents the same characteristics as hypersthenic gastritis—more or less infiltration with embryonic cells, preservation of the chief cells, and decrease or increase in number of the border cells, and glandular proliferation. Associated with this local inflammation is often found general chronic hypersthenic gastritis, more intense in the pyloric region and accompanied by more or less interstitial infiltration. The relation of this form of inflammation to the genesis of ulcer has already been discussed.

The blood-vessels in the region adjacent to the ulcer often show remarkable changes. The process is one of progressive destruction by proliferating endarteritis. The capillaries, the venous radicles, and the arterioles are alike involved, and a thrombus composed of a mass of white cells may be seen plugging the contracted lumen. The wall of the blood-vessel is infiltrated with amorphous matter and with embryonic cells. The endothelium proliferates and the muscular coat undergoes fatty degeneration. In rare cases of infectious origin the clots may contain pathogenic bacteria. There can be no doubt that this devascularizing process is a protection against hemorrhage. But the arterial wall may be softened and may yield to the pressure of the blood before its

lumen is obstructed. The hemorrhage may be venous, capillary, or (nearly always) arterial—slow, continuous, recurring, small, or fatal in a few minutes.

In the majority of cases a plastic peritonitis develops over the apex of the ulcer. The inflammation is circumscribed and productive, the thickened peritoneum being covered by pseudomembrane, often leading to the firm adhesion of the stomach to adjacent parts. Infrequently, the peritonitis extends over a large part of the stomach. The process is a bungling effort at conservatism, offering a protection against perforation, but forming adhesions which impair the motor functions of the stomach, and which, while preventing a rapidly fatal purulent peritonitis, leads to circumscribed abscesses, destruction of an adjacent organ, burrowing of pus, pyemia, cachexia, and usually death.

In about one-half the cases of ulcer the stomach is bound to an adjacent part by plastic peritonitis. On account of the relations of the parts of the stomach most frequently the seat of ulcer, the adhesions, in about seven-eighths of the cases, are between the stomach and the pancreas or the left lobe of the liver. Infrequently, the stomach is united with the diaphragm, the abdominal wall, the omentum, the spleen, or the colon. The adhesions may also be multiple, and often interfere greatly with the churning and the evacuating movements of the stomach. They may also long remain sensitive or may become the seat of stubborn neuralgia.

In about five per cent. of the cases the ulcer perforates the gastric wall, coming in contact with adherent adjacent tissues or producing encysted or general purulent peritonitis.

Perforation after adhesions have been formed is most frequent where the ulcer is located on the posterior wall, near the lesser curvature, or in the pyloric region. But the comparatively rare ulcers of the anterior wall perforate more frequently, and, on account of the free movements of this part of the stomach, nearly always before adhesions have formed. Death follows this accident—rapidly, from shock, or in a few days, from general purulent peritonitis.

The opening in the peritoneal coat is small, varying in size from a pin-head to a pea, usually rounded and clear-cut, but sometimes irregular in shape with ragged edges. The opening is made by gradual erosion or by necrosis and digestion after the peritoneal blood supply has been cut off. Increased intragastric pressure may be the occasion of the perforation.

Perforation after adhesion with the pancreas brings the resistant tissue of this organ into communication with the

cavity of the stomach and its contents. In the base of the ulcer (the perforation often being large) may be seen the reddish-yellow glandular structure, interlined by the grayish interstitial framework of the pancreas. The destructive process may extend into the substance of the gland and form a number of fistulæ, or may open a blood-vessel and produce a fatal hemorrhage.

The liver is much less resistant than the pancreas, and purulent inflammation destroys its substance rapidly, leaving in its stead a cavity containing pus and communicating through a small opening with the cavity of the stomach. The perforation may open a communication with the gall-bladder, the colon, the small intestine, the spleen; with the abdominal wall, resulting in the formation of a gastric fistula; with the diaphragm, through which pus may perforate and invade the organs of the thoracic cavity.

As a consequence of perforation a general or localized purulent peritonitis may be excited. The pathological anatomy is that of perforative peritonitis. Subdiaphragmatic abscess results when the affected area is walled in, and is usually located in the left, but the collection of pus may also be found in the right (rare), hypochondrium, according to the seat of the perforating ulcer.

An abscess may also be formed when the perforation takes place after adhesions, the new tissue of union yielding to the ulceration and the abscess communicating through the small opening with the cavity of the stomach. The subdiaphragmatic abscess may open into the peritoneal cavity, or may perforate the diaphragm, the pleura, the pericardium, the heart, or may open through the lung into a bronchus.

Gastric ulcer either ends in perforation, with its disastrous consequences, or heals, unless interrupted in its course by a fatal accident.

The appearance and the effects of the cicatrix vary according to the size, the form, the depth, and the location of the ulcer. If the ulcer heals only after the destruction of the mucous coat, the depression marking the repaired defect may be small or only discoverable on close inspection. But usually the scar is star-shaped, with a central mass of connective tissue sending out in various directions lines of fibrous tissue of variable length. The central depression may be formed of fibrous tissue developing in the organ or the part to which the stomach has been united by plastic peritonitis. The organ is then deformed by being drawn up in folds, and a funnel-shaped sac is created. The mucous membrane may

be thrown into irregular folds by the contraction of the scar tissue.

Certain deformities interfere in a remarkable manner with the functions of the stomach. The pylorus may be drawn almost against the cardia; the round organ thus formed evacuating its contents with difficulty. Or a band may extend transversely around the stomach and divide it into two cavities united by a small opening. Very infrequently the cardia is obstructed. Pyloric obstruction is common, and may be due to accompanying inflammatory swelling or to the cicatrization of a pyloric ulcer. This is a very grave deformity, and about one-tenth of the deaths due to ulcer are produced in this manner. Gastric ulcer may heal perfectly, or it may leave deformities which impair the functions of the stomach, or which destroy life by inanition and by auto-intoxication.

Clinical Description.—There is no anatomical disease of the stomach the clinical expression of which is more variable than that of gastric ulcer. The clinical history is more frequently the expression of the associations, the accidents, and the complications than of the ulcer itself. The beginning takes its predominant characteristics from the mode of action and the nature of the causes. The evolution is defined by the accompanying gastritis, hyperchlorhydria, hyperesthesia, peritonitis, adhesions, perforation and its consequences, the effects of the deformities and of the inanition.

The anatomical lesion constitutes the danger, and about its origin and evolution gather the conditions, the accidents, and the diseases which reveal its presence. In typical cases the diagnosis presents no difficulty; in atypical cases it may rest upon a probability; the latent form may create no suspicion of its existence. It is not possible at a given moment to say whether the disease will terminate in a perfect cure, in chronic invalidism, or in death. The variable clinical expression embodied in the modes of beginning, of evolution, and of termination makes clear the variable etiology, genesis, pathological anatomy, state of nutrition, and complications.

Ulcer may have no clinical expression, and may run its entire course without exciting the suspicion of the patient or of the physician, ending in complete recovery. This is the completely latent form, the scar being found at the autopsy after death from some other cause. If a complete clinical history could be obtained it would probably be found that the disease had not run its course in complete silence, but that the subjective symptoms were not so severe as to cause the patient to consult a physician, or were considered so com-

mon as to be unworthy of notice, of mention, or of treatment. This form is most common among the poor, and must be ignored in the clinical description.

The subjective symptoms may be suppressed during a part of the course of the disease. These are the purely anatomical periods in the beginning and during the evolution, occurring as breaks in the progress of the disease, or during the period of healing, but more frequently in the beginning. The anatomical periods create no suspicion, are deceptive, and induce a false sense of security.

The period of formation of the ulcer may be latent or anatomical. In this mode of genesis the ulcer develops without a symptom. It may represent a very short period, during which the gastric juice is eating out the circumscribed dead or dying piece of the gastric wall. It may be readily understood why an ulcer, generated rapidly after thrombosis, embolism, or infarct, and unaccompanied by gastritis, by peritonitis, or by a complication, might not be manifested by any subjective or objective sign. The genesis of ulcer in the course of an infectious disease may also be concealed, the gastric symptoms being masked or misinterpreted, or suppressed by the weakness of the organism. Or, again, the beginning may be characterized by a few irregular, indefinite, and, for diagnostic purposes, meaningless subjective and objective signs. This anatomical period of invasion may end suddenly with perforation or with profuse hemorrhage, or may more slowly assume the common clinical characteristics of ulcer—the special pain, the vomiting, the nervousness, the anemia, and the irritation. About one-fourth of all clinically recognizable cases of ulcer develop in this manner.

In other cases the clinical expression of the period of invasion is atypical, and the recognition of the disease is dependent on the results of the examination. This is the physical mode of beginning. The patient complains of discomfort, of flatulency after meals, but has no true pain; at times, possibly, a little nausea and a poor appetite; or there may be only headache, constipation, and soreness in the epigastrium. The subjective symptoms are digestive, but have none of the definite characteristics of those typical of ulcer. The clinical history does not suggest gastric ulcer, which is revealed by the examination; or the examination marks the case as doubtful; and the efficiency of specific treatment or the subsequent developments of the case confirm the suspicion of an ulcer. The physical mode of beginning charac-

terizes the early period of about one-fourth of the cases of simple ulcer of the stomach.

In one-half of the cases the symptoms of the initial period are those of adenohypersthenia gastrica or of hypersthenic gastritis. The clinical description of these diseases has already been given. In the majority of these cases the symptoms of hyperchlorhydria, of hyperchylia, or of hypersthenic gastritis precede the development of those characteristic of ulcer. In a small number of cases the symptoms of ulcer and the accompanying diseases seem to begin and to develop together. These diseases, so often found associated, are, etiologically, points of the same vicious circle. In this mode of invasion the existence of the ulcer may be revealed with certainty from the beginning by the clinical history, standing in clear contrast to the anatomical and the physical modes of development. But in a number of cases, for a longer or shorter period, it is impossible to exclude or to detect an ulcer accompanied by hypersthenic gastritis or by adenohypersthenia gastrica.

The evolution of ulcer after it has become a clinical disease may be slow or rapid. Perforation or hemorrhage may be the first revealing sign, and either of these accidents may prove quickly fatal. The rapidly fatal perforative form is most frequent in girls, who may be thus suddenly taken off in the bloom of youth. The hemorrhage may at once or after several recurrences be fatal; or the patient may recover from the effects of this accident, and the evolution of the ulcer may continue. These rapidly fatal cases are described as acute, but a quick termination by death from a complication should not be considered a characteristic of the morbid process which is in its nature essentially chronic. The quickly fatal cases are preceded by a longer or shorter anatomical period.

The evolution of ulcer may be short, and may end within six or eight weeks in cure by cicatrization. These are the true acute cases; the ulcer beginning at a particular moment (after traumatism, for instance), manifesting itself by typical symptoms and signs, and, under proper treatment, disappearing completely and permanently.

But this is not the usual course, which is slow, chronic, variable, often with exacerbations and remissions, and with certain symptoms predominating and defining various clinical forms of ulcer. The chronic form of ulcer lasts a variable time. The duration may not exceed one or five years, but

frequently the ulcer remains unhealed for fifteen or even twenty-five years. It is the persistence of the anatomical lesion and of its consequences during this chronic course which maintains the clinical unity of the disease beneath its variable play of expression. The distinctive symptoms are the peculiar pain, the hemorrhage, and the vomiting, which are associated with other symptoms occasioned by the functional activity of the stomach.

The early morning, when the stomach is empty after the night's repose, is the most comfortable moment for a patient with ulcer. There is usually, before rising from bed, no pain nor discomfort, and the appetite is commonly preserved. There may, however, be a slight headache, small appetite, or dread of food, and no refreshment from the broken sleep; the patient may be in some cases worn out by pain, nervous, and weak. But the early morning is still the most comfortable period of the day. After breakfast the digestive symptoms begin, variable in the different cases, but in relation with the quantity and quality of food—pain, heartburn, acid eructations, relieved often by the vomiting of an acid fluid mixed with food, or by the passage of the chyme into the intestines.

During the interval between the digestive periods the symptoms are comparatively quiescent, but they reappear on the taking of food. In addition to these, there may be other digestive symptoms, of secondary importance, and due to the state of nutrition and of the nervous system, and to the complications.

The Cardinal Symptoms.—The genesis of the **pain** of gastric ulcer is very complex. That the constitution and the temperament of the patient exert a great influence there can be no doubt. Some suffer intensely; others bear severe pain with indifference, and are not subject to neuralgias. This subjective element explains why in gastric ulcer, with practically the same conditions, the intensity of the pain should be so variable. Much depends, also, on the temporary condition of the nervous system. All conditions accompanied by increased irritability of the nerve-centers sharpen the perception and generalize the effects of the pain. Of these, none are more active than mental and physical fatigue and excitement. Sexual excesses and menstruation have a similar effect. On the other hand, the pain of ulcer is quieted by repose, and in the uncomplicated cases it subsides completely during the night. The means, consequently, adopted to assure mental, physical, and moral repose are important symptomatic remedies.

The pain of gastric ulcer has also an anatomical basis. The nerves are uncovered by the eroding process, and more or less neuritis may be present. The inflammatory infiltration of the walls, produced by irritation or by infection, or by an effort at repair, and the contraction of the newly-formed tissue, compress the nerve filaments. The traction involved in the production of the deformities and the adhesions of parts normally free are other sources of irritation and pain. The peritoneum may become inflamed and exquisitely sensitive, and perforation may occur, with its resulting purulent inflammation, abscess formation, and pus tunneling. The gastric mucous membrane may be hyperesthetic or inflamed, and its secretion may be excessively acid and irritating. The pain is also excited by the chemical, the mechanical, and the thermal irritants introduced into the stomach. The pain of gastric ulcer is thus very complex in its genesis, and, necessarily, very variable in its intensity and in its qualities.

There is no anatomical disease of the stomach in which pain is so predominant as in ulcer. Of all the symptoms it is the most constant, and may be continuous, intermittent, or periodical. It is almost never absent after a meal containing chemical irritants and large enough to distend the stomach. During the interval when the stomach is empty, and particularly at night, it usually subsides; but in some complicated cases (retention, continuous secretion, adhesions, perigastritis) the patient is never entirely free from pain. It may be completely absent during a period of variable length without evident reason, or after a severe hemorrhage. As a rule, the greater the inanition, the anemia, and the nervous exhaustion, the more constant and severe is the pain. The pain may be the only symptom, and, exceptionally, it is scarcely noticeable; but even in the latent form there has usually been slight, irregular gastric pain, considered by the patient as insignificant. It is commonly severe enough to cause the patient to seek relief.

The intensity and the quality of the pain are variable, but when characteristic it is paroxysmal, digestive, severe, and localized. In very old cases (probably with adhesions or contracting scars) the pain may be dull and drawing—a peculiar discomfort like that of gastroptosis. In other cases a peculiar sensation, usually associated with a little pain, is felt, as if the food were arrested and confined to a particular part of the stomach. The description of the patient would seem to indicate a reflex muscular effort to isolate or protect the ulcer. But the characteristic pain of ulcer is paroxysmal,

severe, raw, gnawing, burning, sometimes pulsating, often excruciating.

The spontaneous pain begins in a small area in the epigastrium (Brinton), or in the back (Cruveilhier), and may be as strictly localized and circumscribed as the tender points. But no negative conclusions should be drawn from the diffusion of the pain, as that of complicated gastric ulcer may extend over a large area. The absence of a point of greatest intensity is no evidence against the existence of the ulcer. The epigastric center of spontaneous pain, which may be as small as a silver quarter or as large as half of the hand, is generally located near the median line and immediately below the ensiform process; but it may be further to the right, lower to the left, or higher, according to the seat of the ulcer and to the position of the stomach.

The diffusion of the epigastric pain is greatest when the paroxysms are severe and when the patient is of a nervous temperament. It may be diffused over the whole gastric region, over the abdomen, or over the precardiac region; it may extend to the left or to the right or directly into the back, or through the brachial plexus into the upper extremity. The excitation may be reflected along the pneumogastrics, and may produce dyspnea. The extension of the pain to unusual points may be due to complications involving branches of the pneumogastric, the intercostal, and the phrenic nerves. When, for instance, the stomach is adherent to the diaphragm, and when this muscle or its serous covering is inflamed, the thoracic points of attachment of the diaphragm may be tender, inspiration may be painful, and the trunk of the phrenic nerve, passing obliquely across the scalenus anticus along the posterior border of the sternocleidomastoid, is painful on pressure. In like manner the pain reflected to the right shoulder may be due to adhesions to the liver.

The dorsal spontaneous pain usually appears later than the epigastric. It is also raw, burning, gnawing, and at times horribly severe. As a rule, it is milder than the epigastric pain, but it may exist alone. The most frequent localization is to the left (sometimes to the right) of the two lower dorsal vertebræ; but it may be lumbar, even sacral, or may be located higher up, in the dorsoscapular region. The location of the pain is supposed to be determined by the seat of the ulcer or by a complication of the ulcer. The dorsal pain may be reflected along the intercostal nerves. An extension to the lower extremities is very infrequent.

The pain of gastric ulcer is always greatest during the period of digestion. The distention and the movements of the stomach, the mechanical, the chemical, and the thermal irritation by the food, the superacid secretion, and the increased flow of arterial blood combine to make the period of functional activity more painful than the period of repose. The pain may occur immediately after eating or may be delayed half an hour or more. It increases with the activity of digestion and subsides with the evacuation of the stomach, lasting about two hours, unless cut short by vomiting or prolonged by stagnation or by retention.

A close relation exists between the irritating qualities of the food and drinks and the degree of pain. It is least after a bland albuminous liquid, like milk. Alcoholic, very hot and very cold drinks, spices, acids, and coarse, solid food excite and increase the pain.

The pain of ulcer is increased by the movements of the body and is calmed by repose. Even abdominal breathing may be suppressed in order to keep the stomach still.

According to the seat of the ulcer, the pain may be increased or relieved by certain positions of the body. The special position is constantly assumed by the same patient, and is always such as protects the ulcer from pressure and from the contact of the gastric contents or prevents traction on painful adhesions.

Vomiting, though a frequent symptom of ulcer, is much less characteristic than the pain. In some cases it is the predominant symptom, and if complete and persistent may lead rapidly to very grave inanition. In others it occurs irregularly, or is incomplete, or recurs periodically. Seldom is it absent throughout the course of ulcer, but it may be replaced by acid regurgitations or by nausea. The frequency of vomiting is due in part to the constitution or temperament, some people vomiting more readily than others. Like pain, it is also favored by mental and physical fatigue, and by nervous excitement. But the vomiting which occurs in ulcer is more frequently the result of hyperesthesia of the mucous membrane and of hypersthenic gastritis, and is most frequent after solid and irritating food. In some cases it is due to continuous secretion, or, particularly in the old cases, to pyloric obstruction or to irritable adhesions.

However caused, the vomiting of ulcer (except in retention or in continuous secretion) is digestive. The stomach, if hyperesthetic, may be intolerant of all food, and the food is ejected almost immediately. But, as a rule, it occurs from

half an hour to two hours after the meal, during the height of digestion, when the pain is intense, is produced easily without nausea, and the pain is relieved if the stomach be emptied. Vomiting is usually followed by a burning sensation in the cardiac region, lasting a few minutes; but if the stomach is not completely emptied, the pain is only temporarily diminished. The vomit consists of an acid fluid, mixed with undigested remnants of food, and sometimes a little bile or blood. The vomiting may occur after each meal, or only after large meals containing excitant food. In retention and supersecretion it may take place when the stomach normally should be empty, and the vomit then is characteristic of these two conditions.

Hemorrhage is more frequent during the day than at night, but may occur at any hour. Digestion is supposed to be the most frequent exciting cause, but the blood is often unmixed with food, showing that the hemorrhage occurred when the stomach was empty. The peristalsis and the distention incidental to the functional activity of the stomach, the functional hyperemia and the increased blood pressure, the activity of secretion furnishing a strong digestive fluid, the mechanical action of the churned contents, may certainly be the proximate causes of a hemorrhage. Hemorrhage, however, often occurs during the period of gastric repose, and is occasioned in other ways. Among the proximate causes may be mentioned intense excitement, great effort, vomiting, and straining at stool—all increasing arterial pressure, which bursts the thin wall of the blood-vessel or dislodges the clot from its mouth. Menstruation is also a proximate cause, and often the first sign of ulcer is a gastric hemorrhage occurring during the menstrual period. The hemorrhage may also be excited by coughing, and it is important to remember this when differentiating gastric hemorrhage from hemoptysis. The use of the stomach-tube is contraindicated in ulcer on account of the danger of producing hemorrhage. The blood-vessel may be unplugged by the bacterial softening of the clot. The true cause of the hemorrhage of ulcer is the opening of a blood-vessel by erosion, by necrosis, and by digestion of the devitalized tissue, aided sometimes by increased blood pressure and by traumatism.

Next to pain, gastric hemorrhage is the most frequent symptom, and, if small concealed hemorrhage be counted, it is probable that ulcer never runs its course without this sign. But, clinically, the hemorrhage is noted in only about four-fifths of the cases. The vomiting of blood (hematemesis)

occurs in 30 to 50 per cent. of the cases, but this percentage would be increased if the cases with small quantities of blood in the vomit were included. In the suspected cases the frequent detection of traces of blood in the vomit (not ingested with the meats nor due to the retching) is a diagnostic sign of great importance. But even a large hemorrhage may not excite vomiting, and with modern conveniences may escape the notice of an intelligent patient. A loss of from 50 c.c. to 100 c.c. of blood produces to the layman no perceptible coloration of the stool, and, on account of the foul odor, if not very evident it is not likely to be sought for carefully by the physician. Hemorrhage is observed in about two-thirds of all cases, but it might be detected if diligently sought for in nearly every case. But a single small hemorrhage would be of very doubtful diagnostic value. Hemorrhage may be the first sign of ulcer, but it commonly occurs a number of weeks or months after the pain.

A glance at the rich arterial supply of the stomach, and the relations of the large vessels to the most common seats of ulcer, will explain why this disease should be so frequently accompanied by hemorrhage, sometimes small, frequently dangerous, and not infrequently rapidly fatal. The celiac axis, covered by the lesser omentum and grasped by the lesser curvature, furnishes the stomach, directly and indirectly, with its arterial supply through its three branches—gastric, hepatic, and splenic. These three arteries, through their gastric branches, form on the stomach two complete arterial circuits. The one is formed by the coronary branch of the gastric and the pyloric branch of the hepatic passing between the two layers of the lesser omentum along the lesser curvature. The other is formed along the greater curvature, between the folds of the greater omentum, by the union of the left gastro-epiploic, given off by the splenic, and the right gastro-epiploic, which is a branch of the gastroduodenal artery, given off by the hepatic. The stomach also receives at its pyloric end small branches from the gastroduodenal artery. The splenic, the largest branch of the celiac axis, after meandering along the upper border of the pancreas and giving off the left gastro-epiploic, distributes between the two layers of the gastrosplenic omentum several branches to the greater curvature of the stomach, which anastomose with the branches of the left gastro-epiploic and of the gastric arteries.

The branches of these arterial circuits, and the smaller anastomosing branches, form over the surface of the stomach a network of small arteries, which send branches to the mus-

cular coat, and ramify in the submucous coat, to be finally distributed to the mucous membrane in the form of a network of capillaries covering the gastric tubules and passing up between them to encircle the mouths of the ducts. From this superficial encircling network the blood is taken up by the venous radicles, and is returned through the splenic, the superior mesenteric, and the portal veins. The pyloric, the splenic, and the coronary branches of the gastric are the large arteries most frequently opened. Sometimes it is the right epiploic; the branches of the splenic artery, being distributed to a region of the stomach seldom affected by ulcer, generally escape. The liability to a profuse hemorrhage increases with the depth of the ulcer.

Clinically, gastric hemorrhage may be rapidly fatal; immediately dangerous; profuse and recurrent; and small, concealed, and dangerous on account of the repetitions.

Rapidly fatal hemorrhage occurs in about three per cent. of the cases of ulcer, and is due to the opening of a large artery, usually one of the branches of the celiac axis. Sometimes an aneurysm has previously formed at the weak point of the eroded wall. Suddenly, without warning, the patient becomes pale, weak, anxious; faints, falls unconscious, and dies after vomiting blood, which is very little changed unless the accident happens during digestion.

Death may occur before vomiting takes place. The hemorrhage may be profuse and concealed, but not so rapidly fatal, the stomach consuming fifteen or twenty minutes in filling, the vomiting being rapid and effortless, and followed by collapse, rolling from side to side, delirium, stupor, and death after a short interval of two or three days. The vomited blood is clotted, pure, unless accidentally mixed with the contents, and may be ejected with such force and in such quantity as to fill the mouth, the nose, and the throat. Sometimes, but not often, vomiting does not occur, and the blood is evacuated within twenty-four hours by the bowels, unless death occurs earlier.

A large and immediately dangerous hemorrhage is more common and characteristic of ulcer. The more profuse a gastric hemorrhage, the more likely is it to be due to ulcer. Three or four ounces of blood at a time are frequently lost in cancer of the stomach, and small gastric hemorrhages occur in a number of diseases; but a gastric hemorrhage of from one to three pints is nearly always due to ulcer. The dangerous form is ushered in by the usual signs of severe hemorrhage—pallor, weak pulse and heart, vertigo, great thirst, syn-

cope. The stomach is full and feels warm; the blood rises into the mouth, and large quantities are vomited without effort or pain. The blood is dark and clotted, the appearance varying according to the time that it has remained in the stomach and to the quantity of HCl with which it has come in contact. Immediately after the cessation of the hemorrhage the patient is weak, exhausted, the extremities are moist and cold; he complains of vertigo and of ringing in the ears; the temperature of the body rises because so little blood goes to the surface, but the fever soon subsides; there may be dark spots in the field of vision, even amaurosis; palpitation is frequent; dyspnea after the least effort; he is anxious, and has restless nights and broken sleep. The subsequent course is that of hemorrhagic anemia, which may disappear in one or two months.

The hemorrhage may be profuse and may recur frequently, death taking place in four or five days; or the hemorrhage may cease after occurring intermittently a number of days or weeks, and the patient may eventually get well. The repeated hemorrhages may be due to the progress of the morbid process, to deficient coagulability of the blood, or to the fact that the blood-vessel is opened and not divided so as allow its coats to retract. The blood vomited represents only a part of that lost, the remainder passing into the intestines.

Equally pernicious are the small repeated hemorrhages, usually escaping detection. The anemia is severe, and the emaciation and the cachexia are remarkable, the termination often being death. These little hemorrhages may occur early when the ulcer is eating its way through the mucous membrane, but sometimes later in the anatomical progress of the ulcer, and they may be venous. There is neither hematemesis nor perceptible melena. Traces of blood must be sought for in the gastric contents and in the stools. Infrequently the hemorrhage is slow, and eventually vomiting may be excited by the accumulated blood; the vomit, then, is brownish-black, like coffee grounds, consisting of blood pigment, debris of cells, fluid, often food, and sometimes sarcinæ. This form of hematemesis is most common in the cachectic stage of ulcer and in the retention stage of pyloric obstruction.

Melena may be the only symptom of ulcer. The blood is small in quantity, and is homogeneously mixed with the contents of the bowel, forming a soft, chocolate-colored mass. If a large quantity of blood passes into the duodenum, the stool is tarry, is often blown to pieces with gas, and is exceedingly foul. After nearly every gastric hemorrhage blood may

be detected in the stool, and melena may occur without hematemesis.

In a case of hematemesis or of melena two questions must be answered: Is the hemorrhage gastric? and is it due to an ulcer? But before searching for the location or the cause of the hemorrhage it is first necessary to detect its existence. This is not so easy as might be supposed, and a small hemorrhage may escape the close observation of both patient and physician, as hematemesis and visible melena may not occur. In suspected cases it is always essential to examine both the vomit and the stools by the methods already described for detecting therein traces or small quantities of blood, and also to examine the blood itself for characteristic signs of hemorrhagic anemia.

Having detected blood either in the vomit or in the stools, or a hemorrhagic anemia, search should next be made for its origin. It should not be forgotten that traces of blood may be introduced with the food. Blood found in the vomit may have originated in the stomach, in the esophagus, the pharynx, the nose, the mouth, the respiratory tract, or even the duodenum. The origin in the mouth, the nose, the throat, and the larynx can be detected or excluded by careful inspection; and this examination should never be omitted.

The differentiation of pulmonary and of gastric hemorrhage may be difficult, but can usually be readily made with certainty. The evidence of the patient is frequently worthless. Some blood may get into the larynx during the act of vomiting and may be coughed up. The blood coming from the lungs in slight hemorrhage may be all swallowed, and, being afterward vomited, may present difficulties that are not easy to overcome, especially when the signs of pulmonary tuberculosis and painful digestion or ulcer coexist. This is more likely to happen with women. To make the differentiation, it is best to proceed in a methodical manner. Do the clinical history and the objective examination reveal a disease of the lungs, or of the heart, or of the esophagus, of the liver, or of the stomach? If a disease be detected, the evolution of which is accompanied by gastric or pulmonary hemorrhage, the discovery is strong presumptive evidence of the source of the blood.

The method of beginning, when carefully observed, is of great importance. Hemoptysis begins with tickling in the throat, with cough, and with the expectoration of red blood. Hematemesis begins with the symptoms of internal hemorrhage, and with a feeling of distention and of heat in the

stomach. The warm blood mounts along the esophagus to the throat, and nausea is followed by vomiting.

The signs following the hemorrhage may be conclusive. In hemoptysis the sputum brought up by coughing continues for several days to be bloody, and for a short period thereafter the blood expectorated is red, and does not consist of particles that have accidentally gotten into the larynx. In hematemesis the sputum becomes quickly clear if the mouth and the throat have been freed from blood.

In hemoptysis there is frequently fever; in gastric hemorrhage fever is ephemeral and due to the loss of blood, and the stools frequently contain blood at some time during the following forty-eight hours. Ulcer is an afebrile disease unless it be complicated.

In hemoptysis the blood is red in the beginning, and is mixed with air. Later the sputum may contain both dark and red blood. In hematemesis the blood is nearly always dark, and presents the changes peculiar to the action of HCl.

The differentiation of esophageal and of gastric hemorrhage may be very difficult. The search for the causative disease should first receive attention—cancer, ulcer, varicose, veins, or the rupture of an aneurysm. The use of neither the esophageal nor the gastric sound is permissible. The discovery of a disease productive of passive congestion in the portal system is of differential value. The blood from the esophagus is dark, but not chocolate- or coffee-colored, and is expelled without the effort of vomiting. The blood goes also into the stomach and hematemesis or melena, one or the other, or both, may occur. The clinical history is of most value. There is, in the one case, a history of esophageal pain, located behind the sternum and extending into the back and shoulders, or of stricture. Both symptoms are manifest during swallowing. In the other case the symptoms are located in the stomach, and begin after the food has reached this organ. In all cases of esophageal ulcer there is vomiting.

Blood found in the stools may have entered at any point of the alimentary canal, and it may be impossible to locate the source of the hemorrhage; but the presumption is in favor of its gastric origin when there is no discoverable intestinal disease and where there is a history of gastric trouble. The blood, when in too small quantity to excite diarrhea, is intimately mixed with the fecal matter, and so altered as to be often recognizable only by the chemical tests.

Objective Signs.—The chief positive physical signs of simple ulcer of the stomach, of diagnostic value, are the epi-

gastric and the dorsal tender points. These points are characterized by their localization, sharp limitation, and very great sensitiveness.

The **epigastric point** is located on or very near the median line, close to the ensiform process. The location may vary according to the seat of the ulcer and to the position of the stomach. Consequently, it may sometimes be a little to the right or to the left, or lower down. It is commonly of an area of about the size of a silver dollar, and the location in a given case is constant.

This small, tender area is sharply limited, and does not correspond in form and location with the left lobe of the liver. In some cases the whole epigastric region is sensitive, particularly if the examination be made when the stomach is not empty. The production of the pain, when the stomach contains fluid, by the little successive shocks employed to elicit the splashing sounds is a distinctive feature. The epigastrium may also be hyperesthetic, but with care the small and more sensitive area can be detected and its boundaries can be defined.

The epigastric point is very sensitive, more so than in any other disease of the stomach, where from two to four times the amount of pressure is needed to produce true pain. But in a number of cases the sensitiveness is not so great as to be characteristic, and the sign alone should not be given too great importance. The degree of sensitiveness varies in different cases and in the same case at different times. The epigastric spot is characteristic in about two-thirds of the cases, and may be present when all subjective signs are absent.

The **dorsal point** is about the size of a silver dollar and is located about an inch to the left of the two last dorsal vertebræ. It is sharply limited, but not so sensitive as the epigastric point. Sometimes a second sensitive area coexists, to the right of the spine on the same level, but requires greater pressure to elicit true pain. Sometimes the point on the right is the more tender, and it may exist alone. Two similar points may be located on a level with the fourth and the fifth dorsal vertebræ; but this is not characteristic of ulcer, but is due to reflex excitation of the spinal sensory nerves by the irritable sympathetic ganglia. The lower dorsal point exists in about one-third of the cases of ulcer, and its diagnostic value is variously estimated.

In the same case the degree of sensitiveness of the dorsal point is more variable and the point itself less persistent than the epigastric tender point. Taken in combination with other

ulcer symptoms, the dorsal point is a confirmatory sign of value.

A physical sign of ulcer, much more common than is generally admitted, is a palpable tumor. If the ulcer be recent, it consists only of a defect of the mucous membrane, but possibly it also extends deeper; in either case no tumor will be felt. In old ulcer, however, the edges may be thickened and infiltrated, and if then it be located in the part of the anterior wall accessible to palpation, or in another region made accessible to the fingers by displacement of the stomach, a flat, thin, and tender tumor can be felt. The pylorus may be thickened, or hard and contracted, if the ulcer is located near it. These forms of tumors are not complicated by adhesions, and can be easily fixed on expiration. More common and characteristic are the tumors formed by inflamed adherent organs and by inflammatory exudation. The localized inflammation and infiltration of the adherent organ produces a circumscribed mass, which is hard and is easily defined by the examining fingers. The head of the pancreas, when felt, is deep, immovable, hard, and enlarged; or the mass may be in the adherent left lobe of the liver, and may ascend and descend with respiration in close union with the diaphragm. The ulcer tumor may long remain stationary, or is, at least, not slowly and regularly progressive. It is tender, develops as a consequence of inflammation, and, considered in connection with the clinical history and the symptoms and other signs, may be very important in the diagnosis of ulcer. The age, the stationary character, the tenderness, and the absence of secondary nodules may be valuable in excluding a suspected cancer.

In ulcer of the stomach the state of nutrition is variable. In the mild clinical forms, where enough food is taken and retained, and the loss of blood is insignificant, the strength and the weight may be maintained, and the general appearance may be that of excellent health. But such a state of nutrition is exceptional, and pain, vomiting, hemorrhage, and the insufficient and exclusively liquid diet spontaneously adopted after a certain length of time produce emaciation and inanition. Indeed, in complicated cases, or where little food has been utilized for a long time, the inanition may be fatal. About five per cent. of the deaths from ulcer are due to starvation. An insignificant hemorrhage in this state of extreme emaciation may prove to be a death-stroke. These cachectic cases are more frequent after the fortieth year. Inanition is more frequent, and emaciation may be more pronounced in ulcer than in any other non-malignant disease

of the stomach, and the cachexia may be as marked as in advanced carcinoma.

In round ulcer of the stomach the blood may be normal or it may be diseased; the disease of the blood may be primary, or it may be secondary, and due to inanition and to hemorrhage. The relations of anemia and chlorosis to the genesis of ulcer have already been discussed. The symptomatic blood trouble is always oligocythemia.

A single small hemorrhage may produce a very slight and temporary disturbance; a single large hemorrhage is followed by greater changes, and the phenomena of regeneration of the blood are more marked; repeated hemorrhages, though small, lead in the course of time to very grave oligocythemia. This is very clearly seen in the hemorrhagic form of ulcer, and where there are frequent small and concealed hemorrhages without either hematemesis or visible melena. The great recuperative power of the blood is a distinctive characteristic of the blood in ulcer of the stomach.

After a single small hemorrhage the number of red cells and the percentage of hemoglobin are proportionately diminished, and there is in a few hours a slight increase in the number of the polynuclear white cells, and a few nucleated red cells may appear after a few days. The red corpuscles are all of the normal size. A comparison of the results of the examination of the blood a short time before and after the hemorrhage in a case of ulcer would reveal the occurrence of the hemorrhage.

After a single large hemorrhage, which is almost sure to be accompanied by hematemesis, the hemoglobin and the number of red cells are diminished, the percentage of hemoglobin divided by the percentage of red cells is equal to unity, and the number of lymphocytes and polynuclear white cells is notably and absolutely increased. After a few hours the blood formula begins to change, and in a few days becomes characteristic. The blood is flooded with small nucleated red corpuscles; the common red corpuscles and the hemoglobin increase, but the cell regeneration is faster than that of the hemoglobin, so that the above fraction is less than unity. The number of red corpuscles are gradually recovered, and the white corpuscles soon drop down to their normal proportion and number. The regeneration is greatly prolonged if repeated small concealed hemorrhages occur. A sudden arrest or a fall in the regeneration or in the richness of the blood, respectively, would be a sign of hemorrhage. The blood changes are thus revealing signs.

Repeated small hemorrhages are very frequent in ulcer. They may or may not be associated with a severe hemorrhage. These small and, in themselves, insignificant hemorrhages cause neither vomiting nor coloration of the stools sufficient to attract the eye. Their repetition renders them serious, and a knowledge of their existence would confirm a provisional diagnosis of ulcer. The blood will be found to have a diminished number of red corpuscles, a diminution of the hemoglobin, and the percentage fraction is less than unity; the number of small nucleated red corpuscles is increased, and also the number of white corpuscles. There are sudden changes for the worse, and the recurring little blood crises indicate the small concealed hemorrhages. In all cases of gastric ulcer the blood should be carefully watched. In hemorrhagic oligocythemia there are no signs of degeneration of the corpuscles, no signs of dyshematopoiesis, and no signs of hematocytolysis occurring in the circulating blood. The oligocythemia is due to the loss of blood by hemorrhage.

The anemia of gastric ulcer may be due to inanition. Pain in itself may exert an influence, vomiting may rob the organism of some of its nutriment, but the chief cause of the inanition is voluntary starvation. The person feels best when he eats little and gives the irritable, sensitive organ rest.

The blood formula of inanition anemia is different from that due to hemorrhage. A distinction should be made between the effects of abstinence due to gastric intolerance and a starvation diet. In complete abstinence and in fasting with the exclusion of all but water, in spite of the starved appearance, in the cubic millimeter of blood there is the normal proportion and quantity of red corpuscles and of hemoglobin; but the number of white corpuscles is rapidly and markedly diminished. This fall may go even below 1000 to the cubic millimeter. It is well known that digestion destroys and draws from the general circulation to the digestive tube a large number of the white cells; but the effect of this momentary diminution of the number of the circulating corpuscles is the generation and the entrance into the circulation of an excess of white corpuscles. This is known as digestive leukocytosis. It seems to be a general rule that leukocytolysis is followed by leukocytosis, and this, whether it be the result of using the white cells as phagocytes, as in the infectious diseases, or in absorption and assimilation. In starvation, this digestive and assimilative use of the white cells is suppressed. The absence of the demand

leads to decreased production, and eventually to diminished productive power.

In chronic inanition, the conditions are different. The individual is trying to live on insufficient food. The blood may long maintain itself at the expense of the fat and the muscles, and the percentage of formed constituents in the cubic millimeter of blood may even increase. The patient looks starved, and the corpuscular richness of the blood is a surprise. At a later stage the hemoglobin and the corpuscles all decrease, and may be reduced, in starvation cachexia, to one-fifth of the normal number. An emaciated, weak patient with a normal blood formula is suffering from inanition. The blood in severe inanition-oligocythemia displays signs of degeneration, of dyshematopoiesis, and of hematocytolysis.

The anemia is not always pure, but may be due to the combined influence of inanition and of hemorrhage, and in the complicated cases with pus formation or with retention and fermentation, is partly also the result of auto-intoxication. These mixed forms of anemia may become exceedingly grave. The blood displays the signs of dyshematopoiesis, of hematocytolysis, and of degeneration, in combination and in divers degrees.

The **functional signs** of ulcer are in no wise characteristic, but may possess a certain diagnostic value. In every case where there is good reason for suspecting the existence of an ulcer, the use of the stomach-tube is contraindicated. While it is true that the tube may be employed without accident, the procedure is dangerous, and may excite hemorrhage. If the tube be used in a suspicious case, the throat should be sprayed with cocain, the contents aspirated, the stomach left empty, and the tube withdrawn if the patient should make an effort to vomit. The functional exploration is reduced, as a rule, by the contraindication to the use of the tube, to an examination of the vomit, which, in the majority of cases (70 per cent.), will be found more acid than it should be at the moment in the evolution of digestion when the vomiting occurred. In other cases the acidity is normal or, rarely, less than normal. In nineteen cases, studied by the careful use of the tube, we found pyloric obstruction already present in three cases, which will be left out of consideration on this account. In 11 of the remaining cases there was hydrochloric superacidity, and three of these showed a moderate prolongation of digestion due to supersecretion. In four cases secretion was normal in quantity and in evolution,

and in one there was hypochylia—the time for the examinations being selected so as to eliminate the influence of subnutrition and hemorrhage. In three of the cases with hydrochloric superacidity secretion became normal during the second week of the ulcer treatment. Hayem reports secretion normal in three of a total of 22 cases with no obstruction of the pylorus. The vomit may also show signs characteristic of other diseases, or such as are found in the complications of ulcer. In uncomplicated ulcer the motor function is efficient, there is no decrease of absorption, there are no abnormal bacteriological signs, and no variation in digestive activity, except that resulting from the frequent hydrochloric superacidity. The functional signs may be of some value in confirming the existence of ulcer, or in excluding it, by revealing a complication or another disease. This information may be obtained from repeated examinations of the vomit or by the careful use of the stomach-tube.

Constipation is the rule in ulcer, and the bowels usually require aid in order to prevent fecal accumulation. A few diarrheal movements are sometimes excited by decomposing blood.

Terminations.—Ulcer may terminate in cure, in death, or in chronic invalidism. The oligocythemia and the inanition resulting from ulcer are favorable to the development of infectious diseases, which may prove fatal.

The healing of the ulcer is characterized by the subsidence of the symptoms and objective signs. Of the cardinal symptoms, the hemorrhage is the first to disappear. Hemorrhage proves that the morbid process is progressing, though when it occurs, some parts of the ulcer may be cicatrizing. Vomiting ceases or is accidental and occasional. The pain diminishes, becomes purely digestive, and exists only after solid or irritant food or after physical or mental fatigue. The tender dorsal and epigastric points become less and less sensitive and finally disappear. With the healing of the ulcer disappears also, as a rule, the digestive hyperchlorhydria. The ulcer before completely healing presents a purely anatomical period without signs or symptoms. About one-half of the cases of ulcer recover without leaving an impairment of the digestive functions as a permanent legacy.

The mortality from ulcer is usually placed too high. Some authors state that 50 per cent. of the cases terminate fatally; but the statistics are compiled from cases reported in medical literature. If death from intercurrent diseases (tuberculosis, cancer) be excluded, the mortality of the clinical (non-latent)

forms is about one in seven, and this percentage may be reduced by treatment. Death may be due to perforation (causes death in 80 per cent. of deaths due to ulcer), to severe hemorrhage (two per cent.), or to other complications (five per cent.), such as inanition, hemorrhagic anemia, and deformities.

The remainder of the cases become chronic invalids. The functions of the stomach may be compromised by adhesions or by deformities. The scars may be neuralgic. The sequelæ may give persistent trouble without being incompatible with life. Death from the effects of the ulcer is frequently prevented only by the intervention of a fatal intercurrent disease.

Diagnosis.—The diagnosis of ulcer may be sure, doubtful, or, in a third class of cases, there may be only cause to suspect the existence of this severe disease. Naturally, the anatomical (latent) form goes unrecognized, and, also, during an anatomical period ulcer would be either unsuspected or would be considered cured. The clinical forms are often atypical in their expression. Whoever waits for all the cardinal symptoms—pain, vomiting, and hemorrhage—to be present, with their distinctive features, before making the diagnosis of ulcer, will discover it late, and will overlook entirely a majority of the cases.

The clinical expression of ulcer being so variable, it is difficult to enumerate all the cases which fall under the three divisions—sure, doubtful, and suspicious. "I am inclined to think," wrote Brinton, "that nothing less than a concurrence of the chief symptoms entitles us to pronounce a decided opinion. In other words, unless the pain possess the characteristics attributed to it, and is accompanied by an equally characteristic vomiting, and unless there be evidence of considerable or repeated hemorrhage in the course of the malady, there is no sufficient ground for affirming the existence of gastric ulcer." These requirements are too exacting.

Wherever the three cardinal symptoms are present there can be no doubt. If the spontaneous pain and the tender points are present, with the definite characteristics already minutely described, there should be little need of confirmation by the other signs, such as vomiting, hyperchlorhydria, hemorrhage, and the state of the blood and of nutrition. A large gastric hemorrhage, in the absence of the symptoms of other diseases which might cause it, may also be considered conclusive. There need be no hesitation if, associated with the gastric hemorrhage, are the special pain, the painful digestion, the vomiting, and the objective signs; not all these combined need be present, but one or more, with their typical charac-

teristics. A positive diagnosis can be made in much less than a majority of the cases of ulcer.

But none of the cardinal symptoms may be present in their typical forms, and the symptom-group may leave the case in doubt. The probability increases with the number of more or less characteristic signs and symptoms. Anemia with painful digestion in a girl, presenting traces of blood repeatedly in the stools and in the vomit should excite more than a suspicion. Suggestive signs may also be given by the mode of beginning and the general characters of the evolution of the case. In all the hypersthenic diseases of the stomach the possibility of the development of an ulcer should not be forgotten, and in all the doubtful and suspicious cases the general principles of the treatment of ulcer should govern the medication adopted. "Suspicions which fall far short of a definite diagnosis," declares Brinton, "may be sufficiently important to dictate the whole plan of treatment. By treating these doubtful cases as ulcer of the stomach, we may often cure what we can not diagnose."

It is always easier to make a diagnosis of ulcer at the writing-desk than at the bedside. Nothing would seem easier than the recognition of a disease with symptoms and signs possessing so many distinctive features. But the physician is not consulted after the clinical evolution of the disease is complete, but while the disease is beginning or running its course. The severe gastric hemorrhage may be in the future, and of no possible use; or it may be in the past, and with nothing to reveal its source except the recollections of a man half frightened out of his wits when it occurred. The pain may not possess its distinctive features, and the patient's lack of observation may keep them in obscurity when they exist. Vomiting is a symptom of too many diseases to be of much value. The same is true of hyperchlorhydria, the detection of which may be prevented by the opposition of the patient or by the danger of using the tube. When the diagnosis is not made easy by typical symptoms and signs, a probable diagnosis should be based on a careful consideration of all the symptoms and signs which are present. In some cases the suspicion of ulcer may be so well founded as to demand the subjection of the patient to the inconvenience of a rest-cure; in other cases the walking treatment should be adopted, and the result of the therapeutic test may confirm or destroy the suspicion.

A knowledge of the seat of the ulcer may be of value in forming a prognosis and in imposing a stringent application

of a methodic cure so as to avoid the probable dangers by arresting the progress of the ulcer and by causing it to heal. It may also suggest the probable necessity of surgical intervention for the relief of a constricting deformity. But the rules for locating the ulcer are so untrustworthy as hardly to enable us to make an intelligent guess. After it has formed, it is easy to diagnose a pyloric or a cardiac obstruction demanding surgical treatment. The guides to the localization of the ulcer lead just as often wrong as right, and very little trust can be given them.

If the ulcer involve the cardia, the pain is immediate on swallowing, particularly after a large bolus of solid food. The cardia is very sensitive to the temperature of food. If the sound be passed, a severe pain behind the ensiform process and extending to the upper dorsal spine and shoulder-blades is complained of as soon as the instrument passes the cardia. The stomach is often intolerant, and immediately ejects whatever is introduced into it. The signs of cardiac obstruction are more trustworthy.

If the ulcer is located in the pyloric region, the patient is more comfortable when on the left side, and the pain is increased when on the right side. If the ulcer is adherent to the liver, the pain radiates to the right shoulder. The pain is supposed to begin later than when the food is more quickly brought into contact with the ulcer. Strong, visible, peristaltic waves, and delayed evacuation, which indicate beginning pyloric obstruction, are of more value.

If the ulcer be on the smaller curvature, or near it on the posterior wall, hemorrhage is frequent. The pain is relieved by the sitting posture or by lying on the left side, and is increased by lying on the back or on the right side. The dorsal spontaneous and pressure pain is marked, and is rarely absent if peritonitis (particularly with adhesions) exists.

If the ulcer is on the anterior wall, hemorrhage is rare, perforation is frequent, the pain is relieved by the dorsal position, and is located to the left or lower down than the usual point; a tumor or thickening may sometimes be felt and peritoneal respiratory rubbing may sometimes be detected.

Differential Diagnosis.—Several painful diseases resemble ulcer in their clinical expression, and make it necessary, particularly in atypical cases of ulcer, to search for differential symptoms and signs. When the cardinal symptoms of ulcer are present, with their usual associations and their distinctive features, the case can be nothing but gastric ulcer. But ulcer does not conform its clinical manifestations to the classical

lines laid down in books, and the practitioner will be confronted by difficulties. The diseases most likely to be confounded with ulcer are gastralgia nervosa, adenohypersthenia gastrica, hypersthenic gastritis (which see), cancer, displacement of the stomach with painful digestion, cholelithiasis, and duodenal ulcer.

In both gastralgia nervosa and in ulcer the pain is gastric, and may be paroxysmal and severe. But the gastralgic attacks are intermittent, begin suddenly, and become rapidly intense; are in no constant relation with the taking of food, or with its quantity or quality, or with the evolution of digestion; are unassociated with a disorder of secretion; and the attacks, extending alike through the periods of digestion and of functional repose, are separated by days of normal painless digestion. The neuralgic pain coincident with digestion may be stilled by anodal sedative galvanization, but this is never true of the pain of ulcer. The special pain of ulcer is excited by food, particularly by solids and by irritants, and is digestive; it is relieved by the evacuation of the stomach, but never by pressure, and is increased by movements and calmed by repose. The epigastric and the dorsal points are localized, sharply limited, and persistent. Digestive superacidity is frequently present. In gastralgia there is never hemorrhage, secondary anemia, nor inanition; but there are often neuralgic pains in other parts of the body. It should not be forgotten that gastric neuralgia may be a sequel of ulcer.

Atypical ulcer may be confounded with the digestive form of adenohypersthenia gastrica. The two prominent symptoms of this dynamic affection of the stomach are hyperchlorhydria and painful digestion. Naturally, no doubt can exist when there has been a large gastric hemorrhage or repeated small gastric hemorrhages. The blood signs of hemorrhagic anemia are very valuable in the exclusion of the non-hemorrhagic diseases of the stomach, provided the source of the hemorrhage can be located in the stomach. But ulcer is often manifested by pain and by hyperchlorhydria, the other primary and secondary signs and symptoms being suppressed. The presence of any anatomical signs would at once exclude the dynamic affection, and signs of inanition would be in favor of ulcer. Hyperchlorhydria is not always present in ulcer, as in the other affection under consideration. But the practitioner may be confronted by a condition where a probable conclusion must be drawn from the characteristics of the pain, which may be sufficient

to suggest the one or to exclude the other. Pain increasing with the evolution of secretion and relieved by albuminous foods, speaks in favor of adenohypersthenia. The pain is acid-produced, and is diminished by combining the free HCl. The pain of ulcer is excited directly by the mechanical and the chemical irritation of the food, though hyperchlorhydria may also be a factor of its genesis. The pain of ulcer, being due to combined influences, is never completely relieved by the administration of albuminous foods. Other special characteristics of the pain of ulcer—such as the relation to the movements of the body, to the attitude, etc.—may be present. The epigastric and tender dorsal points of ulcer are present during the period of gastric repose. The causation, the genesis, and the evolution may be in favor of the one or the other disease. In some cases doubt can only be dispelled by time, and in the meanwhile the treatment suitable for the more serious disease should be employed.

The physician is often called upon to make the differentiation of ulcer and of cancer of the stomach. This may be a problem of easy solution, or one that necessitates a close study of the distinctive features of the symptoms common to the two diseases; or a search for symptoms and signs present only in the one or in the other. There may be general features and little peculiarities and associations which speak in favor of a benign or of a malignant process. These minor points are given in the clinical descriptions of the two diseases, and will often be found of more value at the bedside than tables of contrasting generalities. Some of the more important distinctive features will here be brought together.

Cancer is most frequent between the ages of forty and sixty, and is rare before thirty. Ulcer is most frequent between twenty and forty, but is by no means rare after this period. The beginning of the disease before thirty is in favor of ulcer, but it should not be forgotten that carcinoma may rarely occur before the twentieth year.

The beginning of carcinoma is acute, and most frequently without any previous gastric trouble. A disease of the stomach, beginning somewhat suddenly, without appreciable cause, in a man beyond the fortieth year, is circumstantial evidence in favor of cancer.

Ulcer frequently begins insidiously, with predisposing and exciting causes; or else suddenly, with a characteristic cardinal symptom. But it must not be supposed that a diseased stomach does not become the seat of cancer.

The evolution of carcinoma is rapid, progressive, uncon-

trollable. The disease kills in about two years, and the clinical period is about fourteen months. There are no absolute breaks in its deadly march, but under proper treatment there may be periods of improvement. Ulcer is a chronic disease, with periods of quiescence and self-improvement, yielding often, as if by charm, to well-regulated treatment. It may also be acute, but its symptoms and signs are then all the more likely to be characteristic, and an early fatal termination may occur in a distinctive manner from profuse hemorrhage or from perforation, and not from auto-intoxication, cachexia, and inanition. But inanition may also develop rapidly in ulcer, as a consequence of the insufficient diet, or of vomiting, or of hemorrhage. In cancer, also, the small repeated hemorrhages may be very pernicious; but the emaciation often continues in spite of the retention of enough food to mantain the balance of nutrition, because a part is lost by fermentation and by deficient utilization, and because catabolism is excessive. In ulcer, however, inanition and cachexia may be as pronounced as in cancer, and may be the cause of death; but this does not occur until the disease has lasted much longer than the clinical period of carcinoma, unless a complication develops.

Pain presents in the two diseases many distinctive features. Naturally, only the painful forms or periods of carcinoma and ulcer come into consideration here. The pain of cancer is not confined to the digestive period, nor does it develop in relation with the evolution of secretion; it is not excited by the taking of solid food, but its intensity often increases after food is taken which easily undergoes lactic acid fermentation. The pain is most frequently the result of excessive acid-production (butyric and lactic acid) by bacteria, but local peritonitis and painful adhesions should not be overlooked. The pain is sharp, lancinating, agonizing, and persists, and, indeed, often is most intense during the period when the stomach should be empty and at rest. It is well known that stagnation or retention is nearly always present in cancer, and, consequently, the pain is often due to the irritant contents. But the carcinomatous stomach, when empty, may also be the seat of severe spontaneous pain. All these characteristics are in marked contrast with those of the special pain of ulcer, already so minutely described. It is only in exceptional cases that pain on pressure is localized, circumscribed, and so acute as in ulcer.

A profuse hemorrhage, with the vomiting of pure blood, is rare in carcinoma, the vomit commonly being "coffee

grounds" in appearance and mixed with the very acid and sometimes stinking contents. Small hemorrhages possess no differential value, though melena is much less frequent in cancer.

The vomiting of ulcer is easy and digestive, and occurs at the climax of the pain, which it relieves. In cancer, vomiting may occur at any moment, and may be repeated soon after the stomach has been thoroughly emptied, and is usually difficult and accompanied by retching.

A tumor may be felt in either disease, but is very much more frequent in cancer. In the one case a very sensitive inflammatory mass is felt, nearly always firmly adherent to the surrounding parts. The tumor of cancer is frequently mobile, nearly always much less sensitive, and often can be fixed during expiration by the examining fingers. But the mere presence of a tumor is by no means conclusive, and neither is its absence very much in favor of ulcer.

Ulcer and carcinoma can nearly always be distinguished with certainty by the functional and the bacteriological signs. Ulcer has only one functional sign, and this is not a symptom of the ulcer, but of the associated adenohypersthenia or hypersthenic gastritis. Fortunately, this one functional sign—hyperchlorhydria—is never found in cancer except in the very rare cases where it is engrafted on an old ulcer. Sometimes, in ulcer, the hydrochloric acidity is normal, but this is very rarely so in cancer. Exceptionally, hypochlorhydria is found at some period of ulcer; but hypochlorhydria is the rule in cancer. In ulcer there is no fermentation, nor excessive nor peculiar germ growth, unless there is retention due to pyloric obstruction or stagnation from motor insufficiency produced by adhesions. But in these complicating conditions hydrochloric superacidity is the rule, and sarcinæ may be found in large quantity (retention); and there is yeast fermentation, with the formation often of large quantities of gas in the stomach and in the fermentation tubes. The functional and the bacteriological signs of cancer are very different—motor insufficiency, diminished secretion, characteristic bacillary growth, and, chiefly, lactic and butyric acid fermentation. Lactic acid is never formed in ulcer after thorough lavage and the oatmeal test of Boas, but it is so formed frequently and often at an early period in cancer. The functional signs of cancer are persistent; the functional signs of ulcer may change rapidly under the influence of sedative and protecting medication.

The displacements of the stomach (vertical displacement

and total descent) are easily determined by their signs and symptoms. The dislocated stomach may become obstructed or myasthenic, and its mucous membrane irritable or even inflamed. Under these circumstances there may be stagnation, superacidity, and severe paroxysmal digestive pain. The problem, then, to solve is whether the displaced stomach is also the seat of ulcer.

The pain of ulcer, associated with a displacement of the stomach, is often atypical. It is often independent of the quality of the food, solid food being eaten without much discomfort. There may be little difference in the subjective symptoms excited by the digestion of a solid meal and of one composed exclusively of fluids. The pain, which usually occurs late, rarely culminates in vomiting.

The painful forms of the dislocations of the stomach have also, as a rule, a tender epigastric point. The discomfort is increased by movement and is relieved by repose. Consequently, in this particular condition many of the ulcer characteristics are not available, for the ulcer may be present in the dislocated stomach, with stagnant contents, fermentation, and hydrochloric superacidity. In the absence of any of the signs of hemorrhage, the exclusion or the detection of an ulcer may be impossible. But a hypothesis may be built on a study of the tender points. Those of ulcer have already been minutely described. In the painful dislocations the epigastric point is never so sensitive as in ulcer, and is supposed to be due to an irritable solar plexus. The frequent association of the epigastric tender point with tender points to either side of and below the umbilicus makes this opinion plausible. Neurasthenic tender points are also often found along the spine, including the cervical and sacral regions. If only one tender point is found over the abdomen, and is located in the epigastrium, and does not correspond in form and area with the left lobe of the liver; if only one tender point is found in the back, and situated to the left of the two lower dorsal vertebræ—if both these points are very sensitive and persistent, the presence of an ulcer is very probable. In ulcer the stomach is always beneath the tender point. The tender point of gastroptosis is above the lesser curvature, and the pain is not produced until the pressure of the finger is exerted on the gastrophrenic ligament. In some cases, with atypical symptoms and signs, the diagnosis or exclusion of ulcer is a mere assertion.

Cholelithiasis and gastric ulcer often come up for differential diagnosis. A profuse hemorrhage or the detection of a

gall-stone in the feces would be decisive. But it should be remembered that the two diseases may be coexistent.

The attacks of gall-stone colic are periodical, and are separated by intervals when an ordinary mixed diet is borne with perfect comfort. During the attack, however, the functions of the stomach may be reflexly disturbed. In some cases the attacks recur with very short intervals, and with marked regularity. But during the short interval one or more mixed meals may be painlessly digested. The course of ulcer may be broken by anatomical periods, but only exceptionally, and never by short periods of severe manifestations and of complete silence.

The pain of gall-stone colic is independent of digestion, and has its center usually to the right of the median line, and radiates to the right into the back and the right shoulder. There is often a sensitive area to the right of the last dorsal vertebra, and the whole hepatic region may be sensitive. If epigastric tenderness exists, it corresponds in form with the usually unenlarged left lobe of the liver. The gall-bladder, situated about two inches to the right and the same distance above the umbilicus, may be exquisitely sensitive and distended; may produce a visible tumor, and sometimes peritoneal friction may be detected during the up-and-down movements of respiration; and, very rarely, the rubbing together of gall-stones may be felt. During or soon after the attack, particularly when the recurrences are frequent, the right lobe of the liver is enlarged, smooth, and sometimes tender. Very important differential signs are the fever and the jaundice which often accompany the attacks of gall-stone colic. The attack also begins without any fixed relation to a meal, and ends in a manner equally inexplicable by a supposed location of the trouble in the stomach.

The most frequent gastric trouble in cholelithiasis is motor insufficiency, which is not a sign of simple primary ulcer; and gastric secretion may be excessive or continuous. Hyperchlorhydria is about as frequent in gall-stone colic as in ulcer, but it may subside between the attacks without special treatment. Whoever has watched the gastric functional signs in the two diseases will not put much faith in their value as differential signs.

The clinical expression of ulcus duodeni is very variable and indefinite. This utter lack of distinctive features is its strongest characteristic. Its irregular manifestations are very similar to those of atypical gastric ulcer, and it is very seldom that an opportunity, when it presents itself, is utilized to

attempt the differentiation, which may sometimes be made with probability. Like ulcer of the stomach, that of the duodenum may be completely anatomical, or latent up to the moment of a fatal hemorrhage or of perforation. In the rest of the cases there may be symptoms which, on account of their location, characters, associations, conditions, and general lack of distinct meaning, may excite suspicion. The following considerations may sometimes render it possible to make a probable differentiation.

Ulcus duodeni is a much less frequent disease than gastric ulcer; it is more frequent in the male sex; one-tenth of the cases occur before the tenth year; it is common after non-fatal extensive burns of the skin, and is not produced by chlorosis or anemia. The pain of duodenal ulcer in about one-third of the cases is a prominent symptom, and is located where the right parasternal line crosses the right costal border; it may be excruciating, and radiate over the abdomen; it does not entirely subside when the stomach is empty, is not often relieved by vomiting, and may begin as late as two or three hours after a meal and continue during the night. Sometimes the taking of food relieves it, and Chvostek notes that the paroxysm of pain was relieved in one of his cases after the ingestion of a glass of wine. If local peritonitis is present, the pain may be severe and continuous, increased by taking food, and the duodenal region may be extremely sensitive to pressure. A duodenal tender point exists in most of the clinical cases, but a dorsal point is seldom found. Vomiting is infrequent, painful digestion is uncommon, and in a case observed by Leube hyperchlorhydria did not exist. A profuse hemorrhage occurs in about one-fourth of the cases, and may produce both stormy hematemesis and melena, but melena alone is the rule. Icterus is sometimes produced if the ulcer is near the opening of the common duct, with foul, fatty stools. These signs and symptoms differ from those of gastric ulcer chiefly in their relative frequency, or in peculiarities which are only exceptionally present. Fortunately, the treatment of gastric ulcer is appropriate for both diseases.

Complications.—The complications of ulcer are numerous, and some are more dangerous than the disease which has caused them. Anatomically, ulcer is either non-perforating or perforating. At any moment during its evolution ulcer may become cancerous. Before perforation, the only other complications are local peritonitis and hypersthenic gastritis. After perforation there may occur ulceration of the adherent

parts, general peritonitis, abscess and its consequences. The secondary oligocythemia, which may be mild, severe, or grave, has been described among the symptoms.

Engrafted Cancer.—About five per cent. of the cases of carcinoma of the stomach develop in the border or in the cicatrix of an ulcer. "The edges undergo cancerous degeneration or a fungous growth shoots up from the base," wrote Brinton, who was the first to notice this complication. No other disease of the stomach seems to form so favorable a soil for the development of cancer. The ulcer, or scar, seems to be the point of least resistance to the atypical, invading, and unconfined epithelial proliferation. Where cancer of the stomach develops in such conditions, it always selects the border of the ulcer or the cicatrix for its location. On the other hand, the development of a peptic ulcer in the part affected by cancer, or even in the healthy part of a cancerous stomach, has not been observed. The ulcer is always the primary disease in which the cancer has become engrafted.

The diagnosis of the complication during life is most often a happy guess, except where a nodulated tumor can be felt in the pyloric region, increasing in size from day to day, accompanied by uncontrollable and progressive emaciation and cachexia, out of proportion to the amount of food eaten and retained. Hyperchlorhydria is the rule, and is as persistent as that of simple ulcer. The functional and the bacteriological signs may not be distinctive. The detection of secondary nodules in the liver would be conclusive in favor of cancer, and the previous history might reveal the existence of the ulcer which has undergone malignant degeneration. Either the ulcer or the cancer will be overlooked, unless the present signs reveal a cancer and the previous history an ulcer. After the development of the carcinoma, the previously benign clinical evolution becomes malignant and progressive.

Plastic Peritonitis.—A local plastic or productive peritonitis is a very common complication before perforation occurs, and may be considered a conservative process. Ulcer may heal after perforation if a base is supplied by an adherent organ or muscle; death, in the natural course of events, is almost certain, if the contents escape into the general peritoneal cavity. But adhesions do not always form, even after the local peritonitis has long existed; particularly if the ulcer is located on the anterior wall, which makes large excursions with both movements of respiration.

The process then can act conservatively only through the possibly increased resistance against perforation made by the thickened peritoneum. At autopsies, gastric adhesions are found in about one-half the cases where ulcer has existed. Including cases where plastic peritonitis exists without adhesions forming, it may be estimated that simple local productive peritonitis occurs in the majority of the cases of ulcer.

There are no signs or symptoms by which the complication can be recognized. Many of the distinctive characteristics of the special pain of ulcer are in reality due to it, and it is probably present in all cases where the tender points are extremely sensitive, and a dull, aching, deep-seated, spontaneous pain, causing the patient evident anxiety, persists during the period of gastric repose. In some cases peritoneal rubbing may be felt, or respiratory friction sounds may be heard over the seat of the trouble, furnishing the only conclusive and revealing signs. Persistent extension of the spontaneous pain into distant or unusual parts, such as the right shoulder, the lumbar region, and the precordium, should excite suspicion. The occurrence of the complication in a recognizable form is a danger-signal, for the ulcer already extends in depth to the peritoneal layer.

Perforation.—In about four per cent. of the cases of ulcer perforation takes place. According to Lebert, in three to five per cent. of the cases perforation occurs, and is followed by abscess or by general peritonitis, and is the cause of about one-fourth of the deaths from ulcer. According to the statistics of Brinton, which were compiled from autopsies reported by various authors, perforation occurs in every seven or eight cases of ulcer. However, fatal cases are more frequently reported, and the statistics of Lebert are nearer the truth. About one-twelfth of all ulcers are located on the anterior wall, and sixty per cent. of ulcers so located terminate in perforation. But perforation occurs in only two per cent. of all other ulcers of the stomach. We have found 58 cases (35 women, 22 men, and one, sex not given) of latent ulcer scattered through literature, the first manifestation of which was perforation, and in 43 of these cases the ulcer was located on the anterior wall. We have collected 1348 cases in which the situation of the ulcer was given, and find it 923 times on the anterior wall and 425 times elsewhere. Weir and Foote report the proportion as 43 to 17, and Comte as 28 to 15, in favor of the anterior surface, in operated cases. Perforation is about three times as frequent in women as in men (1483 to 461). The cases in

men are scattered all along through life, the average of the collected cases being about 40. Four-fifths of the cases in women occur before the thirty-fifth year.

Perforation of the stomach may take place when the viscus is full or when it is empty. If perforation occurs during distention of the stomach with food and gas, severe pain is the first symptom. The pain is sudden, severe, often agonizing, and it does not cease when the stomach is empty. The epigastric pain is quickly followed by distention of the upper part of the abdomen, which is rigid, and it is accompanied by shock and by anxiety, and sometimes by a subnormal temperature. The pain may be only dulled by an ordinary dose of morphin, but it frequently moderates spontaneously after a few hours. Vomiting is a symptom in more than half of the cases. The pulse is feeble, the breathing is superficial, rapid, and thoracic, and the temperature soon begins to rise. Death will occur from general peritonitis unless an operation is performed, and an operation is demanded as soon as possible after the development of these symptoms in the course of ulcer. When the abdomen is opened gas escapes, but there is no foul odor, though the sour odor of the escaped gastric contents may be perceptible.

The stomach may be empty when perforation occurs, and the pain is not likely to be so severe nor the shock so great, nor peritonitis so general and rapid in its beginning and course, as in the cases where perforation takes place during digestion. Indeed, the peritonitis may be circumvallated if the patient is kept quiet and the stomach empty and still. Recovery may take place without an operation, but it is the rule for the peritonitis to become general after a few days of deceptive quietude, and eventually to take the patient off.

Ulceration.—Perforation may occur after adhesions have been formed. The results of perforation under such circumstances are ulceration, inflammation, and abscess formation in the adherent part, leaving often a fistulous connection with the stomach.

The ulceration which has eaten through the gastric wall invades the adherent organ or part. When the pancreas forms the base of the ulcer,—and this is most frequently the case,—purulent inflammation and abscess formation are infrequent, the tissues of this organ being unusually resistant. The ulcer heals, or eats its way gradually, by molecular death, into the glandular structure or into the excretory ducts, producing thus a pancreato-gastric fistula. The liver may be invaded in the same way; but this organ is less resistant than the pan-

creas, and the ulceration, progressing eccentrically, forms a large cavity, or the process may be walled in by interstitial inflammation. But more frequently a hepatic abscess forms, which is badly drained by its fistulous connection with the stomach. Ulceration may also form a canal of communication between the stomach and the gall-bladder, between the stomach and the intestines, and between the stomach and the external world, through the abdominal wall. The ulceration may extend through adhesions to the spleen, and then commonly produces a splenic abscess. Another and a very dangerous route is by way of the diaphragm, causing, as the morbid process advances, productive or purulent inflammation, and filling the traversed cavities with decomposing pus and gas. In this way the pleural cavity (left), the pericardium, the heart, the mediastinum, and the lungs may be involved. It is extremely infrequent that these parts are reached directly by simple ulceration; but far more frequently through the mediation of a subphrenic abscess. During the extension of the ulceration a blood-vessel may be encountered, and its wall may be eroded. Hemorrhage is the natural result.

The diagnosis of these many complications is made through the signs of ulcer, of local peritonitis, and of adhesions, and through those furnished by the secondary affection of the particular organ or parts involved. The complications may be accompanied by fever, but uncomplicated ulcer is always an afebrile disease. The signs of ulcer and of local peritonitis have already been given; the other diagnostic signs will be given in the paragraphs on abscess and on the sequelæ.

Abscess.—Subphrenic abscess is a very serious, but fortunately an infrequent, complication of ulcer of the stomach. Maydl (1894) has collected in all 35 cases of gastric origin, three of these being caused by carcinoma and 32 by ulcer; some of the cases enumerated by Nowack being thrown out because the etiological diagnosis was doubtful. Brinton (1859) states that "there are about 20 cases of chronic perforative abscess on record." These statistics by no means include all the cases of this complication, for as a clinical entity it is a comparatively recent disease (Leyden, 1880), the first clear clinical description (by Barlow, 1845) having attracted little general attention. Cases (chiefly found at autopsies) are reported here and there in medical literature, but the complication, in its clinical and practical aspects, has been best studied by Barlow (1845), Bouchard (1862), Rigal (1874), Cassy (1879), Leyden (1880), Nowack (1881), and

Maydl (1894). The monograph of Maydl is a masterful presentation of the subject.

Perforation into the peritoneal cavity, according to Lebert, occurs in from three to five per cent. of the cases of ulcer. In some of these, on account of the minuteness of the opening, on account of the degree of intragastric pressure at the moment of perforation, and of the protection afforded by an adjacent part, or other accidental circumstance, the peritonitis is circumscribed, and the pus is walled in and confined to the abscess cavity. Perforation after adhesions have formed is more frequent, and the tissue uniting the stomach to the adjacent part may break down or be perforated, and subphrenic abscess results. The infection may be conveyed along the lymphatics, and the abscess may form without perforation of the stomach. The complication, consequently, is not so very infrequent as the small number of authenticated cases would indicate, and its pathology and its diagnosis should be more generally known, for surgical interference gives the only hope of cure.

In about one-half of the cases the ulcer is located on the lesser curvature and the posterior wall; more rarely, near the cardia or on the greater curvature. The left lobe of the liver, though it be previously bound to the stomach by adhesions, offers little protection against the development of an abscess; and in about one-tenth of the collected cases the stomach was firmly adherent to the pancreas, which formed the base of the ulcer.

For anatomical reasons, the abscess due to ulcer of the stomach is nearly always intraperitoneal, and located in the left concavity of the diaphragm. It is sometimes located in the right hypochondrium, or lower down, and separated from the diaphragm by an intervening organ.

In a vertical section of the body, on about a level with the ninth dorsal vertebra, two oval cavities are formed, surrounded on the surface of the section on all sides by the diaphragm, and separated from each other in the median line by the pericardium, the esophagus, and their associated parts. The oval line of the incised diaphragm is separated from the abdominal wall by the pleural cavities and the lungs. The right and larger cavity of the diaphragm is filled by the right lobe of the liver, and the left and smaller cavity by the fundus of the stomach. As the sections are made successively lower down, the diaphragmatic ovals increase in size and the spleen appears on the left side. Much earlier than expected, the splenic end of the transverse colon and

the splenic flexure appear, and explain why this viscus often forms the lower boundary of the left subphrenic abscess.

The serous covering of the stomach is not complete, and leaves this organ in immediate contact with the diaphragm at two points, whereby an extraperitoneal subphrenic abscess of gastric origin is made possible. One of these points is situated immediately adjacent to the cardia on the side toward the spleen, and the other between the kidney and the suprarenal capsule, the posterior layer of the lesser omentum leaving a small space uncovered.

The boundaries of a subphrenic abscess vary according to its location to the right or left of the suspensory or falciform ligament of the liver, which separates the two diaphragmatic cavities. The abscess in ulcer is seldom situated in the right hypochondrium. The exceptional locations need not be minutely described.

The left-sided subphrenic abscess is nearly always bounded above by the diaphragm. The remaining constituents of its wall depend on the form and the size of the abscess and on the relative location of the organs in the left hypochondrium, which often are displaced or deformed. To the right the abscess is usually bounded by the diaphragm and the left lobe of the liver, but it may extend also as far to the right as the falciform ligament. Below the left lobe of the liver the stomach, the spleen, and also the colon and the lesser omentum, through adhesions to one another and to the abdominal wall, form the lower limit of the abscess cavity, according to the size of the abscess and its extent downward. The limiting wall of the abscess cavity is, to a varying depth, infiltrated with pus, and here and there are erosions and often multiple perforations.

The abscess cavity is filled with pus alone or with pus and gas. The pus is always foul, decomposing, possessing often a fecal odor, when the colon forms a part of the wall. Mixed with the pus is a quantity of granular matter, of false membrane, of necrotic tissue of the limiting organs, of bacteria, and, infrequently, of gastric contents. The gas which is present in two-thirds of the gastric cases may be from three or even four possible sources. In three-fourths of the cases the air enters the abscess cavity through the communication with the stomach. This is always the source when the abscess has resulted from perforation before adhesions have formed, and in cases where the communication was established after the formation of the abscess. The air enters the abscess cavity, in the remaining fourth of the cases, through a communication

with a bronchus. The admission of air then occurs suddenly, and late in the evolution of the disease. The other possible sources are through an external fistulous opening through a secondary perforation of the intestines. In none of the reported cases have germs been found to be the producers of the gas. Gas-forming micro-organisms may form gas in a completely closed abscess, but no case of the kind has been observed in subphrenic abscess complicating ulcer. It is probable that part of the gas in most of these abscesses is a decomposition product.

The complications of a subphrenic abscess are numerous. Perforations into the gall-bladder, the colon, and the pericardium are extremely rare. But pericarditis without perforation is more frequent, and may be dry, exudative, or purulent, the inflammation being propagated by contiguity. In one-seventh of the cases of abscess occurring as a complication of ulcer the lung is invaded and a bronchial communication is established.

The diaphragmatic and the pulmonary layers of the pleura adhere, and purulent pneumonitis and necrosis follow perforation, the abscess of the lung communicating eventually with a bronchus. A circumscribed pneumonia may also be produced by extension from an inflamed pleura.

Possibly, a little more frequently the pleural cavity is perforated, and the contents of the two cavities below and above the perforated diaphragm are the same. Whenever the diaphragm forms a part of the wall of the abscess,—and it is rarely otherwise,—pleurisy without perforation is nearly always present at an early date, and may be pseudomembranous, exudative (with or without blood), or purulent.

The diagnostic signs and symptoms may be divided into two classes—those produced by the abscess and those produced by the primary disease of which the abscess is a complication. Accordingly, the twofold object is the detection of the subdiaphragmatic abscess and the discovery of its cause.

The signs and the symptoms which reveal the existence of an abscess beneath the diaphragm are :

1. The signs and the symptoms of the development of an abscess in the epigastric or the hypochondriac regions.
2. The nature and the evolution of the diseases developing in consequence of the abscess.
3. The position of the diaphragm as revealed by inspection.
4. A cavity containing gas located beneath one containing fluid.

5. Removal, by puncture at different levels, of fluid possessing different qualities.

6. The increased pressure to which the fluid is subjected during inspiration.

7. Certain physical signs.

The subphrenic abscess of ulcer develops rapidly or insidiously. If it be the result of perforation before adhesions have formed, the sudden beginning is marked by severe epigastric pain and tenderness, with more or less shock, and sometimes a rigor and a slight elevation of the temperature. The diaphragm and the upper part of the abdominal wall are held rigid, so as to avoid the pain due to movements. Nausea is common, but vomiting is very infrequent. Hiccup and pain on pressure over the course of the phrenic nerve are sometimes present, and may be due to the diaphragmatitis. The abdomen may be but slightly sensitive on pressure, or may be exquisitely tender, particularly over the region of the peritonitis, where the physical signs of peritoneal rubbing or friction may exist. After a day or two the symptoms become localized; the lower abdomen is no longer distended or tender; but, in marked contrast therewith, the upper abdomen, particularly over the site of the abscess, is swollen and sensitive, and if the pus is in contact with the parietal peritoneum, subcutaneous edema, extending from the hypochondrium into the back, may develop The bowels may be constipated, but, as a rule, move spontaneously or after a laxative if the colon is not involved by the limiting peritonitis.

The beginning may be insidious, particularly where there has been no perforation, or perforation after the formation of adhesions. The spontaneous and the pressure pains of ulcer are no longer localized, but develop over a new, and, for simple ulcer, an unusual, region. The effort to immobilize the parts is evident in the rigidity of the trunk and in the shallow thoracic breathing. There may be slight chills, with increase of the dull or the aching pain and the tenderness, and with slight fluctuations of the temperature. All these symptoms, occurring in the course of ulcer, should immediately excite suspicion. If the abscess be situated superficially there may be a well-marked area of dullness (only pus) where normally it should not be found; or in the usual position on the back, assumed by the patient, the percussion note is tympanitic, and the examination should then be repeated in a sitting posture, and on the side; and it should be observed whether an area of dullness appears where tympanicity before existed (pus and gas). The subsequent course and symptoms are very

variable, partly those of the abscess (emaciation, hectic fever, etc.), but chiefly those of the diseases caused by it, death occurring early or after a long illness.

A very important sequence of diagnostic value is the development of the secondary diseases of the thoracic organs after the abdominal trouble has been in activity for a length of time. This fact fixes clearly the etiological relation of the abscess to the pleurisy, to the pneumonitis, and to the pericarditis. For a time there are no expectoration, no cough, and no physical signs of thoracic disease. The disease of the lungs and the pleuræ develop from below upward. In one series of cases occurs diaphragmatic pleurisy, adhesion of the base of the lung to the diaphragm, pneumonia of the lower lobe, and possibly bronchial perforation with profuse purulent expectoration—no history or signs of bronchitis, of bronchiectasis, or of phthisis. In another series of cases are found an upward-extending pleurisy, empyema, or pyopneumothorax. An ulcer of the stomach, followed or accompanied by the local signs and symptoms of purulent inflammation in either (rarely the right) hypochondriac region, and invasion of the thorax later by purulent inflammation, always most intense at the base, form a sequence strongly in favor of the existence of a subphrenic abscess as a link in the chain of evolution.

The existence of the secondary thoracic disease complicates the condition but makes the diagnosis easier. This is particularly true when the abscess cavity contains gas. "The circumstances of the air being below the fluid in the chest, and the improbability of the pneumothorax being completely separated by a false membrane from a large effusion into the pleural cavity on the same side," was given by Barlow as one of the important signs of a subphrenic abscess. This brings up one of the most important points in the diagnosis—the location of the diaphragm above or below the collection of pus or of pus and gas.

The movements of the diaphragm are often visible, and by inspection the position of this muscle may be located by the line of depression which moves up and down with respiration. In subphrenic abscess the diaphragm is higher on the diseased than on the healthy side of the chest; its respiratory excursions may be very short, or it may be fixed by adhesions or almost completely destroyed by purulent inflammation.

The removal by acupuncture, at different levels, of fluid of totally different qualities, or on the same level but at different depths, may be an important diagnostic sign. But this is an

untrustworthy sign, and every precaution should be taken in order to be sure that one specimen is obtained from above and the other from below the diaphragm. Naturally, if both specimens consist of the same kind of pus, no conclusion can be drawn therefrom. If gastric contents are mixed with the pus, a connection of the pus cavity with the interior of the stomach is demonstrated; but if it be freely connected with the interior of the stomach, the abscess cavity will also contain gas. Sacculated pleurisy and empyema are as frequent as subphrenic abscess, and it is seldom that this double puncture sign can have much differential value.

Great importance has been given by Pfuhl to the relation of the changes of pressure in the cavity to inspiration and to expiration. Jaffé suggests that instead of using a manometer, the changes in the strength of the current running through a cannula be observed. If the flow is greater during inspiration than during expiration, the abscess is in all probability located below the diaphragm.

Certain physical signs may have a differential value, but are, possibly, present only when the cavity contains air. These two signs are amphoric breathing and metallic tinkle and bubbling, heard low down below where the diaphragm is normally attached or is situated in the particular case. The signs gain immensely in value if above them there is a zone of dullness, with no breath sounds nor vocal fremitus.

Before the organs in the thoracic cavity have become involved the diaphragm is pushed up, and moves less during respiration than on the healthy side. There are no abnormal auscultatory signs over the lung if the subphrenic abscess contains no gas; but the region over the abscess may be dull or tympanitic, this latter sign being attributable to the air-distended stomach in contact with the pus. The normal auscultatory signs cease suddenly when this area is reached. If the subphrenic abscess contain much gas, amphoric breathing and metallic tinkle should be heard, and also succussion sounds. The heart is pushed up, but is not much displaced to the right.

After the disease has crossed the diaphragm there may be pleurisy, and there is no physical sign of differential value, unless the abscess cavity contains gas, which will be discovered below fluid; or there may be pyopneumothorax, but without a history of a disease of the lungs to cause it; or, again, the lungs may be perforated, and the expectoration and the physical signs of the purulent pneumonitis, developing from below upward, will be found, in addition to those furnished by the abscess beneath the diaphragm.

The etiological diagnosis is dependent on:

1. The clinical history revealing a particular disease which preceded the development of the abscess, and which is known to be a cause of subphrenic abscess.

2. The location of the abscess.

3. The contents of the abscess.

Maydl divides the etiological diseases into 12 groups: Stomach (35 cases), intestines (13 cases), appendicitis (25 cases), echinococci (17 cases), traumatism of the abdomen (18 cases), liver (20 cases), perinephritis (11 cases), metastatic abscess (11 cases), open wounds (6 cases), caries of rib (3 cases), thoracic diseases (9 cases), and other causes (11 cases). From these statistics it will be seen that about one-fifth of all cases are of gastric origin, and of the gastric cases about 95 per cent. result from ulcer. The signs and the symptoms of these causative diseases should be sought for in the clinical history and in the examination.

The location of abscess due to appendicitis, to perforation of the intestines, or to disease of the gall-bladder is almost without exception on the right side. On the contrary, the abscess due to ulcer occurs almost exclusively on the left. Maydl gives 29 on the left, two on the right, and two on both sides. In about one-third of the cases caused by appendicitis and intestinal perforation the abscess cavity contains gas. The presence of gas is noted in five-sevenths of the gastric cases collected by Maydl.

A subphrenic abscess, preceded by gastric symptoms, but by none of the characteristic signs or symptoms of the other etiological diseases, located in the left hypochondrium and containing gas, is due to a perforating ulcer of the stomach.

Sequelæ of Ulcer.—Clinically, ulcer terminates in a cure, in death, or in chronic invalidism. Considered as an anatomical process, ulcer is either perforating or non-perforating. The non-perforating ulcer may heal with or without deformity, with or without adhesions. Perforation may occur before or after adhesions have formed. If it takes place before adhesions unite the stomach to adjacent parts, there results either general peritonitis or local peritonitis and abscess, which latter may eventually produce a fistula. If adhesions already exist, the result may be healing, or a fistula, or abscess with its many possibilities. Briefly, the sequelæ of ulcer are: (1) Adhesions; (2) fistulæ; (3) deformities.

1. *Adhesion* of the stomach to adjacent parts is a very common sequel of ulcer, forming either before or after per-

foration. There are no characteristic symptoms or signs by which the condition can be recognized. It must not, however, be supposed that adhesions produce no symptoms, for the pain and the uncomfortable and indefinite sensations excited by the distention and the peristalsis of the stomach, and occurring after the ulcer is healed, should be attributed to adhesions.

2. *Fistulæ* are rare sequels of ulcer. The most common are gastro-intestinal and gastro-external. The diagnosis of the latter presents no difficulty. If the cavities of the stomach and the colon be united, the result is likely to be starvation, on account of the small intestine being excluded from digestion; but this exclusion is not complete unless the orifice of communication is large. Fecal vomiting, should it occur, would excite suspicion of the existence of the fistula, and the character of this fecal matter might give some clue to its involving the colon or the small intestine. If the perforation is in the colon, the appearance in the stomach of a colored or easily recognizable fluid introduced into the colon establishes the diagnosis, the introduced fluid being removed from the stomach by the tube. Or the rapid passage of the contents of the stomach into the colon would arouse suspicion. Communication with the bile-passages would open a way for the constant entrance of bile into the stomach.

3. The *deformity* resulting from the loss of substance and from the contraction of the scar affects either the wall of the stomach or one of the two orifices. The deformities may exist in many varieties and degrees, some compromising the motor function very little or not at all and others producing stagnation, retention, or even complete obstruction.

The deformities of the wall are difficult to recognize during life, and the signs and the symptoms are the result of the insufficiency of the motor function. Stagnation or retention without either myasthenia or supersecretion can be due only to obstruction or to a deformity of the gastric wall. The two common deformities of the wall are the hour-glass constriction and the shortening of the lesser curvature so as to bring the cardia and the pylorus closer together. The latter condition is not likely to be even suspected during life, but it may be a cause of stagnation or even of retention. The hour-glass stomach, or the division of the viscus into two communicating cavities by a constriction, may be congenital or inherited, or may be due to dissociated and unequal contraction of the muscular layer. This "physiological" division of the stomach into two cavities by muscular contraction

only occurs during violent peristaltic effort, and in the otherwise normal stomach is a symptomless condition. It is not so with the cicatricial hour-glass stomach, the constriction being persistent, the communicating orifice sometimes very small, and the mechanical work very imperfectly done. The signs of this deformity which aid in its recognition are not very characteristic. If the abdominal wall be thin and flaccid, inflation of the stomach would make the peculiar deformity evident on inspection and palpation. If the communicating orifice be small, the peristaltic auscultatory intragastric sounds would locate the constriction, which should not be confounded with the spurting pyloric sounds heard at another point. The alternate relaxation and contraction of the pylorus, which may sometimes be felt during digestion, would aid in the recognition of the pylorus. Another sign is obtained during lavage of the stomach. The cardiac cavity having been thoroughly washed out, during the continuance of lavage the clear water suddenly becomes mixed and cloudy with gastric contents. To detect and to control this sign, which can only be present under special conditions, the lavage should be made during gastric digestion, and when the water comes away clear the contents of the pyloric cavity should be massaged through the communicating orifice and removed by the tube. The existence of the constriction may be demonstrable by the balloon sound of Kuhn. Still another sign is one first noted by Jaworski. This is an inability to withdraw anything from the stomach when splashing can be demonstrated in it. The fluid and the gas to which the splashing is due are in the pyloric division. The physical signs of this deformity should be sought for in all cases of motor insufficiency following ulcer.

Obstruction of the pylorus and cardia are the most frequent and serious sequels of ulcer. These diseases are described in another chapter.

Prognosis.—Ulcer is a dangerous disease, and the prognosis should be very guarded. Brinton gives the high mortality of 50 per cent., which includes death during the course of ulcer from intercurrent diseases. About 20 per cent. of the clinical cases die, but this high mortality can be reduced one-half by consistent methodical treatment. The death-rate of ulcer is still less, for the purely anatomical form is not rare, and gets well, or is counted only when fatal, since the latent fatal cases are included in the mortality reports. The death-rate of all cases of ulcer which receive treatment is less than ten per cent.

Ulcer is a treacherous disease, and, however mild it may be, a guarded opinion should be given. The course of the disease is beset with danger and with possibly fatal accidents.

The probability of healing increases with the reparative power of the individual organism. Healthy nutrition, a vigorous nervous system, rich blood, and good circulation are favorable influences. The prognosis is also better when the patient gets early and proper treatment. Unfortunate circumstances may render it impossible to carry out a strict rest and diet cure, and the mortality is more than doubled by food of bad quality, by forced work, and by unhygienic quarters.

The probability of death increases with the depth of the ulcer. The blood-vessels increase in size from the surface of the mucous membrane to where they empty into the loops of small arteries; and the greater the depth of the ulcer, the more likely is the hemorrhage to be profuse. Another danger-point is reached when the ulcer has eaten down to the peritoneum, and opens up the possibility of adhesions, abscess, perforation, general peritonitis, and other complications.

The location of the ulcer, where this can be made out, is another guide. Ulcer of the anterior wall is very prone to perforation, and when near the orifices of the stomach may leave obstruction as a sequel.

The preëxistence of advanced hypersthenic gastritis makes the future dark. This disease, be it the cause, or the accidental association, or the result of ulcer, is an obstacle to healing. The persistence of symptoms and signs due to this form of gastritis, in spite of proper methodical treatment, is an unfavorable sign. Grave is the outlook when stagnation or retention exist in consequence of myasthenia or of obstruction; and chronic invalidism is a very probable sequel when a tumor due to perigastritis can be felt.

Treatment.—Something can be done toward the cure of ulcer by the removal or the treatment of its cause. The destructive lesion persists after the influences that generated it are no longer active. The predisposing and the exciting causes should receive attention, both with a view to preventing a recurrence of the disease and to securing conditions essential to the cicatrization of the ulcer. The anemia should be treated and the hyperchlorhydria should be controlled.

The indications to be met by the methodical treatment of ulcer are very clear, and fortunately the remedies employed are usually sufficient and effective. Ulcer, as soon as it is

recognized, should receive immediate and careful attention, for it is a disease which endangers life, and delays and compromises are inadmissible. Expectant treatment is a great blunder, for simple ulcer in a vigorous adult has no "innate tendency to heal," and the grave accidents which are too often the heralds of coming death occur somewhat regardless of the age, the constitution, and the general state of nutrition. The treatment must in every case be immediate, methodical, and sufficiently vigorous to be effective. A compromise may mean death or irreparable injury.

The general indications are: (1) To protect the stomach and the lesion against all sorts of irritation; (2) to give the stomach functional rest; (3) to maintain nutrition; (4) to treat the gross symptoms; (5) to treat the complications.

The means employed to meet these indications are hygienic, dietetic, and medicinal. The treatment, being also protective, consists in part in avoiding injury. The remedies employed are chiefly medical, for it is the business of the physician to treat ulcer. But some of the accidents, complications, and sequelæ may demand the aid of the surgeon.

To obtain the cicatrization of an external open wound, protection against irritation and immobilization are essential, while every precaution is being taken to secure asepsis. The ulcer should be kept clean, still, and protected, and its walls should be maintained in close apposition. Intragastric asepsis is, of course, a chimera, and fortunately unnecessary, inasmuch as simple ulcer of the stomach progresses by molecular necrosis without pus formation or excessive inflammatory reaction. But the walls of both the non-perforating and the perforating ulcer do get infected; and an effort to keep the contents of the stomach sweet, and the ulcer as free as possible from purulent infection, would not be unnecessarily made.

It is only an ordinary precaution to keep the nose, the mouth, and the throat clean, and to prevent the swallowing of purulent discharges. But the surgical principles of more importance in the treatment of ulcer, but capable of only imperfect application, are immobilization and protection against mechanical, chemical, and thermal irritation.

Partial immobilization of the stomach can be obtained by rest in bed and by keeping the organ perfectly empty. This is a favorable condition for the healing of the ulcer, although the ulcer may cicatrize under conditions much less favorable. All convulsive movements of the diaphragm—coughing, laughing, hiccup—should be controlled; and all effort or straining should be avoided. This course of conduct not only aids

healing, but also gives some protection against such accidents as hemorrhage and perforation. Rest in bed is ordinarily an essential part of the treatment, and the so-called "walking treatment" of ulcer is an almost inadmissible compromise. As a rule, it is not necessary to keep the patient in bed longer than two or three weeks, after which time the cure may be completed by a free use of the lounge. Where there is either perigastritis or hemorrhage, or signs and symptoms indicating that the ulceration is progressive, absolute rest in bed should be enforced. This rest cure is beneficial in many ways.

The mechanical effect of rest is evident, and is favorable to healing; it is also a precaution against hemorrhage, perforation, and the extension of perigastritis. But a no less important result is diminution of the needs of nutrition. The body requires much less nutriment when in a state of repose than when in activity. This saving represents so much less digestive work to be done, and makes it easier to maintain the balance of nutrition on the small quantity of unirritating food which it is possible, in ulcer, to give without injury.

The mucous membrane of the stomach is exposed to many forms of irritation. In ulcer, the influence of extremes of temperature seems to be very injurious. The food and drinks should be neither very cold nor very hot, but as nearly lukewarm as possible without being distasteful. Nothing mechanically irritating should be swallowed, and this necessitates a fluid diet, combined or not with completely digestible and nutritive solids in a fine state of subdivision. All drugs must be excluded which irritate the mucous membrane. The diet should contain nothing that is chemically irritating, nor which remains long in the stomach. The secretion of the stomach, on account of its frequent excessive acidity, in ulcer is a chemical irritant; so nothing should be given that excites secretion, and the hydrochloric acid should be at once combined or neutralized, and never left free.

The protection of the stomach against injurious irritation, then, can only be secured by a very strict and proper diet, by the exclusion of most drugs, and by the neutralization of the free acidity or by exclusive rectal feeding. Proper alimentation is of the greatest importance, and the diet will be first considered.

Milk is not only a valuable food, but also exerts a remedial influence in ulcer. Where milk is well borne, the dietetic treatment in the beginning is an exclusive milk cure. This

food, when properly given, fulfils admirably the requirements of a nutritive remedy. It is unirritating, combines rapidly the secreted HCl, does not excite secretion excessively, being normally digested without leaving more than a mere trace of HCl free, is rapidly and easily evacuated by the stomach, and, when given fresh, sweet, and sterile, does not ferment in the ulcerated stomach free from stagnation or retention. But unfortunately an exclusive milk diet is a starvation cure, and is only capable of maintaining the balance of nutrition of the inactive body for a short time, when given in the quantity admissible in ulcer. The cure should always be begun with absolute rest and a milk diet; but the development of inanition, which constitutes one of the dangers of this disease, should be most stubbornly fought.

In some cases the stomach is intolerant, or, on account of a hemorrhage or of fresh perforation, must be given complete functional rest. This perfect repose may be secured for a short time by rectal feeding. The best forms of nutrient enemata are given in the section on General Medication. All the water and a little of the nutriment required by the organism may be introduced in this way. But rectal feeding should also be employed to supplement the insufficient milk diet.

The quantity of milk given by the mouth during the twenty-four hours should not exceed two quarts, and should be perfectly sweet and unskimmed. The emulsionized fat is not objectionable, and adds to the nutritive value of the milk. For the first day, a glass may be slowly sipped every three or four hours, or until it is certain that the milk is well borne even a smaller quantity may be tried. After a few days one glass should be given every two hours, eight glasses being administered during the twenty-four hours. No more than one glass of milk should be given at a feeding, and the intervals between the feedings should be long enough to allow the stomach to become empty. It is a good plan to have an interval of four hours after the fourth glass, so as to avoid accumulation and distention of the stomach. This leaves an interval of eight hours between the last evening and the first morning glass. The four pints of milk possess a nutritive value of about 1200 C., and are not quite sufficient to maintain the balance of nutrition while the patient is resting in bed. One or two tablespoonsful of milk-sugar may be added to the first glass of milk taken in the morning—it is both a food and a laxative. The emaciation which soon begins may be diminished by protecting the body against loss of heat, and by rectal feeding.

The exclusive milk diet is continued until four or five days after the pain and vomiting subside, and after the tender points become less sensitive. The nutritive value of the fluid diet should then be increased by additions to the milk. The quantity of water in the four pints of milk is sufficient, and should not be increased. Two or three times a day a small quantity of a thoroughly cooked preparation of wheat should be stirred into the milk. If this is well borne, a few days later instead of one of the glasses of milk a cup of meat broth, with a beaten egg slowly stirred in while the broth is not hot enough to coagulate the albumin, may be given. Instead of the egg, two tablespoonsful of freshly prepared meat powder or a teaspoonful of "Somatose" may be used. A liquid diet of this sort should be continued until there is no longer any digestive discomfort, the changes in the diet being suggested not by the lapse of weeks and of days, but by the improvement in the symptoms and the signs. Tender meats and the preparations of cereals and fresh butter should be gradually added, and the patient should be held for several months on a mixed, non-irritating diet. The chief components of this diet are milk, eggs, cereals, butter, and the tender meats. Later may be added vegetables, in the form of purées; but acid fruits, alcoholic drinks, rich foods, condiments, and sweets must long be avoided.

The diet is sufficient to nourish the body, provided repose is at the same time enforced and the intestines are healthy, and the work of the stomach is greatly reduced.

Two other conditions demand attention and may be treated by the same remedies—the hydrochloric superacidity and constipation or diarrhea. The inanition, the hemorrhagic anemia, and the favor shown the stomach tend to correct the hyperchlorhydria, the best remedy for which, if it be a reflex sign of the ulcer itself, is to keep the stomach empty for twenty-four hours; but the hydrochloric superacidity is frequently an expression of an associated hypersthenic gastritis. No remedies are then so beneficial as alkalies in large doses, and the contents of the stomach should be kept nearly neutral by their free administration. The three most useful alkalies are the bicarbonate of soda, calcined magnesia, and prepared chalk, and these may be advantageously combined. From two to four drams of the soda should be given during the twenty-four hours, and enough magnesia and chalk should be combined with it to control the constipation or to check a temporary diarrhea. The quantity for the twenty-four hours

should be divided so that one powder is given at the end of each feeding. This neutralization and laxative treatment contributes to the relief of the symptoms, favors cicatrization by combining the free HCl, which is a cause of the chronicity of ulcer, promotes the early and easy evacuation of the stomach, and does not lessen the utilization of the milk. The alkaline cachexia is an imaginary danger, and the neutralization method should be employed wherever there is hyperchlorhydria, which is also, in a degree, controlled by small doses of the extract of belladonna. Instead of these alkalies a Carlsbad cure may be employed, or a glass of Célestins vichy, with enough sulphate of soda to regulate the bowels, may be given every morning.

When the diet is digested without pain, or heartburn, or eructations, or hyperchlorhydria, all drugs should be kept out of the stomach, except possibly nitrate of silver and subnitrate of bismuth, which are supposed to exert a favorable influence on the cicatrization of the ulcer.

There can be no doubt as to the value of nitrate of silver. The greatest benefit will be obtained from its employment when there is either functional or inflammatory hyperchlorhydria, or hyperesthesia of the mucous membrane. It often relieves rapidly the pain and the vomiting. There is no reason to believe that it exerts a direct specific action on the ulcer, but clinical experience proves the value of the drug in all hypersthenic states of secretion which are not due to the stagnation or the retention of irritant contents. One-fourth to one-half of a grain should be given daily, with an intermission after two weeks. This dose, dissolved in a tablespoonful of distilled water, should be given every morning twenty minutes before the first feeding. In rebellious cases, with no history of hemorrhage, a solution of nitrate of silver (1 : 2000) may be used to douche the cleansed stomach. The procedure may be repeated every three days, and controls the excessive secretion of the hypersthenic gastritis better than any other remedy.

The bismuth treatment has some very warm advocates. The pure subnitrate may be recommended in very large doses. The indications for its employment are the same as those for the nitrate of silver; but it is of less value. One or even two drams should be given daily in a single dose, preferably in the morning half an hour before the first feeding. The patient should lie on the back for ten minutes, and then on the right side, with the fond hope that the bismuth may fall into the ulcer as a protecting deposit. Small

doses do no good; the large doses may not constipate, but should not be given for a long period. Fleiner uses the bismuth in large doses, and introduces it through the stomach-tube, after lavage in the early morning before breakfast. Two to three drams of pure subnitrate of bismuth are mixed with a glass of water, and the mixture is allowed to flow into the stomach. The patient then occupies various positions—on the back, on the right and left sides, upright, and in the knee-elbow position. After five or ten minutes the water is withdrawn through the tube. The procedure is repeated daily until the pain disappears, and then the intervals are made longer. Fleiner has used from ten to twenty ounces of bismuth before interrupting the treatment, without producing symptoms of poisoning. The bismuth forms a protecting coating over the ulcer, stimulates granulation, controls the excessive secretion, and Fleiner claims that it is also a hemostatic. Bismuth has given us most excellent results, without employing lavage. There is little gained by withdrawing the small quantity of water (only a small part of the bismuth is recovered after ten minutes), and it would seem that the stomach is already clean, or may be sufficiently cleansed by a glass of Carlsbad water administered one hour before the bismuth is given. We ordinarily give a daily dose of one or two drams for a week or ten days by the mouth, suspended in half a glass of water, and are somewhat timid about using the tube.

The hemorrhage of ulcer is small or profuse. The most essential part of its treatment is absolute rest in bed, and a continuously empty stomach. No food, or fluids, or ice, or medicine should be given by the mouth. The administration of tannin, acetate of lead, subsulphate of iron, and other locally acting hemostatics is irrational. It is not probable that they will come in contact with the part on which they are intended to act. Worthless, also, is ice, being virtually the same thing as the administration of lukewarm water, and the only certain effect is the dilution of the blood and the prevention of its coagulation. The stomach, when it is still and empty, and strongly retracted, is in a most unfavorable condition for the continuance of the hemorrhage. As a last resort, in a continuous hemorrhage lasting for several days (very rare in ulcer but more frequent in carcinoma), the stomach may be washed out with ice-cold water and left empty.

The first object is to arrest the hemorrhage, and the treatment is different when the hemorrhage is small and when it is

large. Absolute rest and mental quietude are essential conditions. These being secured, an ice-bag is kept over the stomach to cause it to retract, and a hypodermic injection of morphin is given to quiet the circulation and to diminish peristalsis. If the hemorrhage continues, ergot should be given hypodermically. This drug contracts the arteries as large as the radial, and strengthens the tonic contraction of the stomach; but its action on the muscular fiber of the stomach is feeble in comparison with its action on that of the uterus. Clinical experience demonstrates the value of the drug in checking gastric hemorrhage.

If, as is usual in ulcer, the hemorrhage be profuse, and the foregoing remedies are not effective, ligatures may be placed around the thighs and drawn tight enough to obstruct the return venous but not the outward arterial circulation. Enough blood may thus be withdrawn temporarily from the circulation to check the hemorrhage and to give time for the clot to form. The dammed-up blood should be allowed to reënter the general circulation slowly.

The hemorrhage being arrested, collapse may demand immediate treatment. The head and the body being placed on a level, the extremities should be raised and the blood massaged out, and compressing bandages should be applied. A pint of a warm physiological solution of common salt (0.6 per cent.) should be introduced into the rectum and its escape prevented. Another pint of the same solution should be injected into the subcutaneous cellular tissue, the points of election being the lower angle of the scapula and the outer part of the thigh.

The heart should be aroused by hypodermics of camphorated oil and ether. Transfusion might be tried to prevent immediate death, in case the necessary apparatus be at hand. For three or four days after the cessation of the hemorrhage rectal feeding should be used exclusively. A little milk may then be given cautiously and tentatively by the mouth, and gradually oral may be substituted for rectal feeding.

The pain is nearly always relieved by the appropriate treatment of the ulcer. Morphin hypodermically, or the extract of opium in suppository, may be required. A very good pain-relieving combination is that of codein, extract of belladonna, and calcined magnesia.

Vomiting may be an obstinate symptom, and if not relieved by rest, by diet, and by bismuth or nitrate of silver, the stomach should be given complete rest for from twenty-four to forty-eight hours, and oral feeding should be resumed under

the soothing influence of the Winternitz compress. Vomiting occurring at the climax of the painful digestive paroxysms is prevented by controlling the pain.

The medical treatment of perforation should be begun without delay, the object being to localize the resulting peritonitis. The patient should be kept absolutely still in bed, and a full dose of morphin should be given hypodermically. The morphin must be repeated in full doses at short intervals, so as to immobilize the digestive tube. By rectal administration it is difficult to keep the patient mildly and continuously narcotized. Ice-cold applications should be placed over the abdomen. The ice-bag is usually too heavy to be borne. The coil of rubber tubing with a continuous flow of ice-water through it is light and convenient. Often cold is not bearable, and the Winternitz compress should be substituted for it. Shock following perforation may demand the hypodermic employment of camphorated oil, ether, and strychnin. Food and water during the first four or five days should be given exclusively by rectum, and the first nutrient enema should not be administered until the digestive tube has virtually been put in splints by the free use of opium. The best and most rational treatment is immediate surgical intervention.

Local peritonitis before perforation should be treated on the same principles, as it may be either purulent or plastic. But the treatment, which is exclusively medical, is much less energetic. Absolute rest in bed, cold over the epigastrium, enough opium to relieve pain and to quiet peristalsis, and exclusively rectal feeding for a few days are the chief points of the management. The stomach should be given complete rest for as long a period as the state of nutrition will permit.

Painful adhesions give a good deal of trouble, and nothing is likely to prove efficient except the gentle influence of time. Counterirritation over the stomach and on either side of the dorsal spine may be tried, and prolonged use of the compress sometimes gives relief. Precautions should be taken to prevent the formation of a drug habit if anodynes are prescribed. Many of the complications and sequelæ can not be treated by operation. Perforation converts ulcer into a surgical disease; abscess must also be treated with the knife; and obstruction of the pylorus or of the cardia may require an appeal to the surgeon for relief.

Some Ulcer Cures.—1. *The Rest and Carlsbad Cure.*—This ulcer cure is very popular in Germany, and has been carefully formulated by von Ziemssen and Leube. Von Ziemssen does

not enforce absolute rest in bed, but forbids physical and mental overexertion. The abdomen is protected by a flannel bandage. Milk, tender meats, milk- and cereal-thickened soups, and white bread are permitted. In combination with this régime must be used systematically a solution of artificial Carlsbad salts in hot water. Two to four drams (one to two heaped teaspoonfuls) are dissolved in a pint of hot water, and the whole is drunk slowly in four portions, fifteen minutes apart, and finished half an hour before breakfast. One or two stools must follow, and, according to the effect produced, the quantity of salts must be increased or diminished. As a rule, one heaped teaspoonful is sufficient after a day or two. If the hydrochloric acidity be very high, a glass of Giesshübel, Bilin, or Vichy may be given at night. Three or four weeks are usually required to cause all symptoms to cease and the ulcer to heal. The Carlsbad salts are now stopped, but the glass of mineral water is continued for a few weeks. Von Ziemssen claims that the cure of the ulcer is due to the salts and to the large quantity of hot water. The highly acid contents are neutralized, the acid fermentation is checked, the stagnation is relieved by complete daily evacuation of the stomach, and a daily movement of the bowels is obtained. There is no doubt that the Carlsbad cure, methodically employed, reduces rapidly, after the first few days, the hydrochloric superacidity, and relieves the constipation. But in ulcer there is neither myasthenia, nor obstruction with stagnation, nor retention and fermentation; or, if these complications be present, the water cure is strongly contraindicated. Carlsbad salts should not be used if there be anemia.

Leube conducts the cure with the patient in bed, and all movements requiring effort are forbidden. Hot compresses are kept over the stomach during the day, and a Priessnitz compress at night; or, in case of hemorrhage, ice-cold applications are substituted. From a glass to a glass and a half of Carlsbad water (Mühlbrunnen) in which from a half to one tablespoonful of artificial Carlsbad salts has been dissolved is given daily one hour before breakfast. But in the beginning the diet consists of the Leube-Rosenthal meat solution, either in bouillon or in milk—both lukewarm. After two or three weeks, pigeon, chicken, purée of potatoes, thickened soups, and bread are permitted for another week, and a normal diet is gradually resumed.

2. *The Rest and Rectal Feeding Cure.*—This method is very efficient, and, among others, has been recommended by Wil-

liams and by Donkin. The patient is kept quiet in bed, and no nourishment is given by mouth. Every three hours an enema (one ounce of beef tea and two ounces of milk, or three ounces of milk—Donkin) is given. After two weeks the same food is given by mouth, and the number of rectal feedings is gradually diminished. This is essentially a starvation cure, and in many cases is unnecessarily severe, and the inanition increases the danger. The ulceration may progress, and a hemorrhage in this condition of emaciation and weakness may be a very serious menace to life. The composition of the best rectal nutritive enemata are given in the section on General Medication. We often employ exclusive rectal feeding for a few days (seldom longer than a week) when the hyperchlorhydria and pain are obstinate, when the motor function is impaired, when a hemorrhage has occurred, and when perigastritis is present. The functional rest and the retraction of the empty stomach both promote healing.

3. *The Milk and Neutralization Method of Debove.*—Debove employs an exclusive milk diet and alkalies in sufficient quantity completely to neutralize all the hydrochloric acid secreted. The milk may be given in small quantity, repeatedly, or in large quantity three times a day. Two to two and one-half liters daily are sufficient to maintain the balance of nutrition, and this quantity should not be exceeded. A glass of pure undiluted milk is given every two hours during the sixteen hours while the patient is awake. A powder or cachet consisting of ten grains of bicarbonate of soda and three grains of prepared chalk is given every hour between getting up and going to bed, and a dose should be given at night if the patient should awake with pain. Or three glasses of milk may be given three times a day. During the three hours succeeding each meal the alkaline powder is given every half hour, and every hour during the remainder of the day.

Under the influence of this treatment the pain and the vomiting rapidly cease. The Debove meat powder, after a week or two, is added to the alimentation, giving at first one ounce a day. A little later cereal powders prepared in the same way may be used.

When the pain and vomiting have been absent several weeks, the alimentation is increased by following the table of digestibility arranged by Leube, and 15 grains of bicarbonate of soda, and the equivalent of three grains each of prepared chalk and calcined magnesia, according to the state of the bowels, are given every half hour during the three hours following the three meals.

This method is rational, and is sometimes very effective, even where severe hypersthenic gastritis coexists; but the complete neutralization of gastric acidity is unnecessary for so long a period in the majority of cases. In practice it is an excellent routine method, but the routine treatment of any disease of the stomach is not commendable. The remedies should be selected which in the particular case best fulfil the indications.

Surgical Treatment.—In certain emergencies the advisability of an operation must be considered. An appeal to surgery may be made on account of the gravity and the uncontrollable evolution of the ulcer, on account of a complication, or on account of a resulting deformity.

Ulcer may be grave on account of the severity of one or more of its cardinal symptoms—viz., pain, hemorrhage, or vomiting. It can seldom happen that either pain or obstinate vomiting should demand surgical intervention, but an operation should be considered a means of possible relief, and a justifiable procedure when the pain and vomiting are accompanied by progressive emaciation and loss of strength and by intolerance of rectal feeding. Repeated hemorrhages or a very large hemorrhage may be treated by operation, the bleeding vessel being ligated or cauterized; or, if the location of the ulcer is favorable, excision may be better practice; or gastro-enterostomy may be performed after the arrest of the hemorrhage. The best surgical treatment is made clear only after the stomach has been exposed to view, provided the strength of the patient will permit the surgeon to carry out the best plan.

An ulcer may be grave on account of its prolonged course and resistance to medication or on account of its location. An ulcer located on the anterior surface and accompanied by local plastic peritonitis should be treated by excision and closure, or possibly by attachment of the stomach with sutures to the anterior abdominal wall—as a prophylactic measure against perforation into the general peritoneal cavity. If the ulcer is unhealed (hemorrhages) and is obstructing the pylorus, it might be well to perform pyloroplasty or gastro-enterostomy. When ulcer is chronic and obstinate, and is accompanied by hypersthenic gastritis, gastro-enterostomy secures rapid evacuation of the stomach and the control of the excessive secretion—two conditions which are very favorable to the healing of the ulcer and to the cure of the gastritis. Doyen reports 21 successive gastro-enterostomies

without a death, but the mortality is 50 per cent. in Billroth's clinic. Comte reports 1 death in 12 gastro-enterostomies for ulcer, and 6 deaths in 17 cases in which the excision or resection of the ulcer was done. (Collected cases.)

Perforation converts ulcer into a surgical disease. Perforation may produce a fistula, or an abscess in an attached (adhesions) organ, or a subphrenic abscess, or general peritonitis. An abscess imperatively demands the use of the knife. In 34 reported cases, 15 recoveries have been obtained by operation (Maydl, Comte, Beck, Abbe, Weir, McCosh); but without an operation recovery is an accident. When the ulcer is situated on the posterior wall or on the lesser curvature, an abscess is the usual result of perforation; but when the ulcer is situated on the anterior wall perforation occurs, as a rule, into the general peritoneal cavity. While recoveries have been reported from perforating ulcer without operation (the stomach probably containing no food at the time of the perforation), yet it may be laid down as a rule of conservative practice to operate whenever perforation has occurred into the peritoneal cavity. The operation should be done as early as possible; the perforation should be closed by suture, either from without or from within the stomach (after or without excision of the ulcer); the peritoneal cavity should be thoroughly washed out and purulent exudate removed, and after-drainage should be invariably employed. Pariser has collected 43 operations for perforating ulcer with 11 recoveries, Comte increases the number to 65 operations and 19 recoveries, and Weir and Foote more recently reported 23 recoveries in 78 collected operations. The following is their interesting table:

Time of Operation.	Recovery.	Death.	Mortality.
Under twelve hours . .	14	9	39 per cent.
Twelve to twenty-four hours	4	13	76 " "
Over twenty-four hours .	4	28	87 " "
Not stated	1	5	
Total	23	55	71 per cent.

The conclusion might be drawn from these statistics that surgery rescues about 29 per cent. of these patients from death; but the statistics are based on reports and not on practice. The truth, were it known completely, might greatly alter the percentage. But we think that it is a good rule of practice, for the guidance of physicians, to arrange for an operation by an experienced surgeon as early as possible after the occurrence of perforation.

Subphrenic abscess is a complication which imperatively demands incision and drainage. Without operation this complication is invariably fatal. Of ten cases operated by free incision and drainage, three (Scheurlen, Debove, Rendu) recovered.

Ulcer may leave, as a sequel, gastric fistula, cardiac or pyloric obstruction, or a deformity producing retention. Gastro-enterostomy is the proper treatment of the latter condition, or adhesions should be broken, and multiple pouches, which have resulted from constrictions, should be connected. Gastric external fistula should be laid open down to the points where it perforates the parietal peritoneum, and packed so as to favor healing from the bottom. The treatment of obstruction of the orifices is discussed in another chapter.

CHAPTER III.

THE NEOPLASMS OF THE STOMACH.

The neoplasms of the stomach are malignant or benign. The benign tumors are very rare, and, being curiosities of the dead-house, they are of very little interest to the physician. Lipoma of the stomach is usually multiple, is either submucous or subserous in origin, and consequently may form prominences on either the mucous or the peritoneal surfaces. These encapsulated and sometimes lobulated new growths are ordinarily about the size of a pea or nut, and produce no alteration of the mucosa or of the peritoneum, except the slight nutritive changes due to pressure. Fibroma or fibromyoma may develop toward the peritoneum, or toward the mucosa, forming a polyp. They are ordinarily about the size of a cherry, occur most frequently in old age, and are most commonly located on the anterior wall and in the pyloric region, and may consequently produce obstructive stagnation or retention. Lymphadenoma is rare, but unlike lipoma and fibroma it may ulcerate; this lymphatic disease, be it neoplastic or diffuse, is always associated with the same affection of the intestines; sometimes the liver and spleen are involved, and the rapid cachexia is accompanied by leukemia. Polyadenoma is a benign epithelial neoplasm, which may accompany chronic proliferating glandular gastritis, and which may undergo cancerous transformation.

The malignant tumors of the stomach are sarcoma and carcinoma. Sarcoma of the stomach is very rare, but secondary sarcoma of the stomach occurs more frequently than does secondary carcinoma. Sarcoma of the stomach presents the same histological characteristics as sarcoma of other organs. We have been able to find only 43 cases in literature. It is most frequent between the ages of fifteen and thirty-five, is nearly twice as frequent in men as in women, and it may suppurate, ulcerate, produce hemorrhage, and cause perforation. Its gastric symptoms and its functional and bacteriological signs are the same as those of cancer. There is oligocythemia, and there may be leukocytosis (polynuclear) as in cancer; but lymphemia is more frequent. The spleen is always enlarged, metastases are frequent and may be accessible to excision and the microscopical examination of a specimen. Sarcoma of the stomach, when it is diffuse, may convert the stomach into a stiff viscus and the pylorus into a rigid canal, producing incontinence instead of retention. Both primary and secondary intestinal sarcomata seldom produce obstruction.

CANCER OF THE STOMACH.

The stomach is one of the favorite sites of primary carcinoma, which is a malignant disease, progressive in its evolution, and fatal in its termination. As a result of modern diagnostic methods, cancer of the stomach may often be surely and early recognized, and a more favorable opportunity is thus offered for radical and palliative surgical treatment. By medical treatment also life may be prolonged and the suffering alleviated. On account of its frequency, malignancy, and the great importance of its early diagnosis, both to the physician and to the patient, the disease should receive careful and minute study.

Frequency.—The number of deaths due to cancer of the stomach varies in different localities, at different ages, and in the two sexes.

If reports are to be trusted, cancer of the stomach is very rare in Turkey, in Egypt (Griesinger), and in parts of South America (Heizmann). It is much more frequent in Switzerland, in Normandy, and in the region of the Black Forest than in other localities of Europe (Antenrieth, Häberlin). In Vienna (Nedopil) there are annually four deaths from cancer in every 5000 inhabitants, and of these one is due to

cancer of the stomach; and the mortality percentage due to cancer is 3.2 per cent., and to cancer of the stomach 0.8 per cent.

The mortality from cancer of the stomach is twice as great in Switzerland as in Vienna and Berlin, one person in every 2500 dying annually of it (Häberlin).

From the statistics of Bryant (official), extending over a period of ten years from 1884–1893, we obtain the following figures:

City.	Population.	Average Number of Deaths Annually.	Average Number of Deaths Annually from Cancer.
New York, . . .	1,628,151	39,943	865
Philadelphia, . .	1,022,355	21,708	487
Baltimore, . . .	437,613	9,120	226
Boston, ...	432,752	10,273	304
San Francisco, . .	313,000	5,979	202
New Orleans, . .	246,021	6,771	152
Total, . . .	4,079,892	93,794	2,236

In these large cities 2.38 per cent. of all deaths are due to cancer, and Häberlin and Bryant have shown that this percentage is slowly increasing from year to year. For New York city the percentage is 2.8 in 1896, or 1 death from cancer to 1697 inhabitants. The average yearly death-rate from cancer in the six cities mentioned is 1 to 1825 inhabitants. From 25 per cent. to 40 per cent. of all cancers are primary cancers of the stomach. Häberlin gives 41.5 per cent.; d'Espine, 42.4 per cent., and Virchow, 35 per cent. About one per cent. of all deaths are caused by cancer of the stomach.

Cancer of the stomach is a rare disease before the thirtieth year. In 1150 autopsies performed on old men, Greenfeld found in nine per cent. that death was due to cancer of the stomach. Less than three per cent. of the cases occur before the thirtieth year, more than two-thirds of the cases between forty and seventy, about one-fourth between fifty and sixty, one-sixth between thirty and forty. Nearly one-half of the cases occur between the ages of forty and sixty. Mathieu (1884) collected from literature 32 cases before the thirtieth year. Wilkinson and Widerhofer observed congenital cases. Cullingsworth and Kaulich have reported cases occurring in infancy. Cancer of the stomach is almost unknown before the fifteenth year, but from this age the chances of dying from it increase with each decad.

About twice as many cancers occur in women as in men, and this preponderance is chiefly due to the frequency with which primary cancer attacks the uterus and the female breast. A compilation of statistics from many sources shows that in women cancer of the stomach is a little more frequent than cancer of the uterus. Cancer occurring in man, in from 40 to 50 per cent. of the cases attacks primarily the stomach; in women only in from 20 to 30 per cent. Bräutigam and Häberlin give the proportion as 6 or 7 to 5; Brinton gives 9½ to 7; and Fox, 8½ to 8. Louis, Valleix, and Lebert claim that cancer of the stomach is slightly more frequent in women. Statistics may be produced to support the contention in favor of the predominance in either sex, and the generally admitted predominance in man is so slight as to be of little moment to the clinician.

Etiology.—The causation of cancer is unknown. Riches, poverty, season, country, city, hard mental and physical work, and inactivity exert no perceptible influence.

Clinical observation claims heredity as a predisposing cause. Remarkable instances of the persistence of the disease in members of the same family through several generations are on record, and cancer is not so frequent that the recurrences can be plausibly explained as accidental. But the reference to inherited influences often means nothing more than a confession of ignorance, and heredity is losing more and more of its domain each day as knowledge increases. Lebert reported that seven per cent. and Häberlin claims that eight per cent. of the cases are hereditary.

Long-continued and repeated irritation is always stated to be a predisposing cause. Some show of reason may be given this contention for cancer in some of its localizations, but on such grounds it would be difficult to explain the genesis of cancer of the stomach. A stomach constantly irritated escapes as often as one which receives better treatment, and one of the characteristics of cancer is its development at an advanced age in a stomach which has previously given no signs of disease. Cancer is a disease which has its favorite sites, and develops exclusively in certain tissues, some of which are remarkably well protected against mechanical and chemical irritation. In this respect it acts like a germ disease. A bacillus has been reported as its cause, and some observers have attributed the disease to sporozoa, or to sporozoa-like bodies, which develop in the epithelial cells. But it is generally admitted that the so-called sporozoa are degenerate cells, or represent endocellular changes in atypical epithelium.

Carcinoma may generally be conveyed from one surface to another with which it is in contact, and metastasis, such as occurs in pyemia and other infections, is frequent. But the metastasis of carcinoma seems to be a cellular transplantation, for the secondary neoplasms consist of the same cell as the mother neoplasm, wheresoever the secondary growths appear. The cancer cell grows and lives like a parasite, and it is hardly possible for a germ to cause the production of cells of a particular kind in organs where these cells do not exist. That the germ of cancer grows in the epithelial cell and imparts to it a malignant reproductive activity is an admissible hypothesis.

Pathological Anatomy.—Anatomically, cancer of the stomach is a disseminating new growth, consisting of a stroma whose interspaces are filled with cylindrical or atypical epithelium. The disease is essentially a malignant epithelial invasion.

The growth of carcinoma, beginning at a single point in the mucous membrane, is best studied along its borders. Proceeding with the microscopic study of the cut through the zone of dissemination toward the center of the new growth, it will first be noticed that the epithelial proliferation is confined within the glands and limited by a basement membrane. The epithelium next pushes out budding projections, over which the basement membrane disappears, and leaves the epithelial cells in direct contact with the very fine and newly-formed stroma, which is usually infiltrated with small round-cells. These projections unite across the tissues separating the glands, and extend into the submucosa and the deeper layers, where cancer cells collect in nests; or the neoplasm develops in lines along the lymphatic vessels, and the bundles of connective tissue and of muscular fibers.

In the formation of the neoplasm two tissues are chiefly concerned. The periglandular connective tissue is infiltrated with small cells and cell nuclei, and out of it is formed the stroma which is to serve as a framework. Contemporaneously with the development of new connective tissue new blood-vessels are formed (vascularization), and in some cases the blood-vessels are very numerous. The characteristic feature, however, is the unconfined epithelial proliferation. The starting-point may be the epithelium about the necks of the glands, and the cancer is then cylindrical-celled; or it may be the epithelium of the fundus, when the epithelium of the new growth is atypical. The new cells stain intensely, and typical and atypical nuclear growth and division (karyokinesis)

is very active. The ducts of some of the glands may be lined with several layers of cylindrical epithelium. The fundus of the glands is usually lined with one layer of new cells, but here and there may be seen two or more layers, and the lumen of some of the glands may disappear. The epithelial cells invade the extraglandular and interglandular tissues, and form here and there nests of cells. Accordingly as the stroma or the epithelial cells predominate, the cancer is hard or soft.

Hard cancer, or scirrhus, is the most common anatomical variety of cancer of the stomach. According to Brinton, it constitutes 72 per cent. of the cases. Hard cancer begins almost without exception in the pyloric region, and infiltrates all the layers of the gastric wall without producing any marked prominences. The wall of the stomach is hard and thick, inelastic, and non-retractile. The pylorus is often converted into a rigid and incontinent tube. The hard, flat mass may be located chiefly on the anterior or posterior wall, or, more rarely, may convert the whole stomach into a hard, rigid tube, little larger than the cecum. The mucous membrane in the area of the tumor is often ulcerated. The ulcer is superficial, with very low receding edges, and the base is smooth and scarlike or ragged and fibrous. The thickened, indurated wall cries under the knife, and is sometimes white and glistening, with but little milky fluid; and is sometimes yellow, streaked, or spotted with hard, white, fibrous tissue. All the layers may be eventually replaced by the carcinomatous stroma, with only here and there a few epithelial cells, not resting on a basement membrane and arranged along the coarse fibers of connective tissue. Scirrhus may be confounded with chronic ulcer or with interstitial gastritis. The differentiation may be made by comparing the evolution; the extension along the lymphatics; the presence of epithelial cells in the submucosa and muscular layer, unconfined by a basement membrane; cancerous nodules in the peritoneal coat, and metastases in other organs. The hard cancer, beginning almost without exception in the pyloric region, destroys and replaces the components of the gastric wall by a compact connective-tissue stroma inclosing a few epithelial or giant cells and cancer nodules and very sparingly supplied with blood-vessels. Ultimately, the stomach is contracted, rigid, and functionless, and the pylorus is obstructed or gaping.

There are two varieties of soft cancer—adenocarcinoma and medullary carcinoma. The first variety is cylindrical-celled, and grows from the epithelium lining the necks of the glands

and covering the surface of the mucous membrane. The epithelial cells constituting medullary carcinoma are small, irregular, atypical, embryonic, and resemble the chief cells of the gastric glands.

Adenocarcinoma is a fungoid, vascular, malignant neoplasm morphologically characterized by a delicate, infiltrated, fibrous framework inclosing cylindrical cells so arranged as to form irregular tubules. The favorite location is the pyloric region and the pyloric ring, originating in the epithelium lining the pyloric glands, and, rarely, also in the cells lining the excretory ducts of the glands of other regions of the stomach. The tumor presents a soft, fungous, red mass, studded with papillæ. The blood-vessels are numerous and often become irregular in caliber, degenerated, and obstructed. Subsequently, the growth of the neoplasm is accompanied by interstitial hemorrhages, necrosis, ulceration, and small hemorrhages into the cavity of the stomach. On section, cancerous juice exudes, and the soft surface consists almost exclusively of epithelium. The tumor grows along the surface and extends very slowly to the deeper layers. A microscopic cut through the zone of invasion reveals irregular cavities lined by cylindrical epithelium resting directly on a framework of connective tissue infiltrated with small round-cells. Sometimes the cavity is filled with epithelium atypical in form and degenerated, the appearance being ultimately very much like that of medullary carcinoma. The glandular arrangement, seen under the microscope, may suggest simple adenoma. But the malignant growth forms a tumor consisting chiefly of cylindrical epithelium without a basement membrane, and invading the layer of the gastric wall beneath the mucous membrane. The formation of cancerous nodes and the occurrence of metastasis are also met with in this variety of malignant disease.

Medullary carcinoma is a papillomatous malignant neoplasm, often ulcerated, consisting largely of small atypical epithelial cells. It grows with great rapidity and invades extensively the glandular system. Like all the anatomical diseases of the stomach, it is more frequent in the pyloric end, but occurs in other parts of the stomach with much greater frequency than does adenocarcinoma. At the autopsy it most often presents an ulcerating tumor, with edges exceedingly irregular in height and thickness, occasionally here and there smooth, but more often rugged, with a base varying in appearance accordingly as it is formed by the submucosa or by the muscular or peritoneal coat. A section of the wall

usually reveals fatty degeneration, hemorrhages, and necrosis. The epigastric, portal, celiac, retroperitoneal, inguinal, and left supraclavicular glands are usually enlarged, and cancerous nodules and cells are strung along the lymphatics in the gastric wall. Microscopically, the epithelial invasion is widespread, extending in the wall of the stomach and involving all its coats. The embryonic chief-like cells are often found within the bundles of muscular fibers and collected in spindle-shaped nests between them.

Either form of soft cancer may undergo colloid metamorphosis, and produce what is often described as a distinct variety. Medullary cancer undergoes this gelatinous transformation or degeneration more frequently than does adenocarcinoma. At the autopsy a tumor appears; commonly as a general thickening of the gastric wall, the inner surface presenting ulcerated, somewhat transparent prominences studded with small, slimy granulations. The outer or peritoneal surface is nodulated. Microscopically, the neoplasm consists of a grayish, fibrous framework, with the irregular interspaces filled with gelatinous matter and with the granular remains of epithelial cells. From the invasion zone may be obtained cuts presenting the microscopic characteristics of the two varieties of soft cancer.

The mucous membrane lining the stomach which is free from the cancerous growth always presents microscopic lesions of an inflammatory and degenerative character. The histological changes are most commonly those found in catarrhal and interstitial and atrophic gastritis, and are most probably due in part to stagnation of the contents and to the influence of the malignant neoplasm on nutrition. The relation of the gastritis to the cancer is not known. In some cases the gastritis undoubtedly precedes the carcinoma, just as carcinoma may begin in the edges of an ulcer of the stomach, or it may be preceded by hypersthenic gastritis. It is possible that these diseases form favorable soils for cancer. But the gastric mucosa, as a rule, undergoes the same pathological alterations when cancer affects another organ, such as the breast. The microscopic changes are those which have been already minutely described under chronic catarrhal gastritis, accompanied by more or less interstitial inflammation and degeneration or transformation of the peptic glands. The cylindrical surface epithelium is ordinarily preserved, but it may be lost when the mucous division of the mucosa is greatly infiltrated by small round-cells and leukocytes. Many of the cylindrical cells of the

surface and of the ducts (mouths) of the glands are converted into beaker cells and ciliated columnar epithelium. The border cells of the peptic glands disappear, and the chief cells are replaced by the cylindrical epithelium which lines the mouths of the glands. The peptic glands are thus transformed into mucous glands; or the fundus of the peptic gland may be destroyed by degeneration and interstitial gastritis, and the cylinder-cell lined ducts elongate and enlarge, and may eventually extend to the depth of the submucosa. Wandering leukocytes and eosinophile corpuscles lie scattered among the infiltrating round-cells. The motor insufficiency results from the cancerous infiltration of the muscular layer in the region of the neoplasm or is due to pyloric obstruction. In the latter case the muscular coat may become hypertrophied. In no other anatomical disease of the stomach is the muscular coat so early, profoundly, and generally affected. Consequently, motor insufficiency is an almost constant sign of carcinoma. The alterations of the mucous and muscular coats produce corresponding and characteristic functional and bacteriological abnormalities.

In the majority of the cases of carcinoma of the stomach adhesions to adjacent organs occur before death. Exceptionally, when the disease is already advanced, the stomach remains free from adhesions, and the tumor may be moved about in the abdominal cavity. The absence of adhesions is most frequent in the cylinder-celled variety. Unfortunately, adhesions are often extensive, and constitute a serious obstacle to operative interference, forming between the stomach and the liver, pancreas, colon, diaphragm, spleen, omentum, abdominal wall, and other contiguous parts.

The method and dissemination of carcinoma is strongly in favor of the parasitic nature of the epithelium which enters into its constitution. The neoplasm may spread by continuity, by contact or inoculation, or by the lymphatics and veins. By continuity of tissue and growth the neoplasm extends in the wall of the stomach to the esophagus and duodenum, and invades adjacent organs over the bridges made by adhesions. Detached peritoneal nodules may transplant the malignant growth to other parts of the peritoneal cavity. The most common method of propagation is along the lymphatics originating in the primary and secondary deposits. The abdominal lymphatic glands are nearly always enlarged, and, less frequently, the inguinal and left supraclavicular glands. Cancer may also be disseminated by the blood, and may thus reach the liver, and from this organ, or directly through the

thoracic duct, it may be carried to the lungs, skin, and other distant organs. The secondary deposits retain the morphological characteristics of the primary carcinoma, and are most frequent in the liver, where they exist in about one-third of the cases. The size of the metastatic growth is in no constant relation with that of the primary carcinoma of the stomach.

Perforation occurs, on an average, in about six per cent. of the cases. The statistics of various authors range from four to ten per cent. Perforation is the result of ulceration or of gangrene, and opens most frequently into the peritoneal cavity. The perigastritis may be walled in and purulent, the subphrenic abscess usually forming in the right hypochondrium. Perforation may also occur through adhesions into adjacent solid or hollow organs, producing an abscess or a fistula. A fistulous communication of this kind is most frequently established with the transverse colon, and sometimes externally through the abdominal wall.

About 70 per cent. of cancers of the stomach involve one of the orifices, and 30 per cent. are diffuse or confined to the body of the stomach. It is commonly reported that 50 per cent. to 60 per cent. of cancers are pyloric. But the situation of the tumor at the autopsy does not make its point of origin clear, and Israel rightly maintains that cancer, as a rule, does not originate in the orifices, but that its extension is arrested there. In 40 cases Boas found that the neoplasm began on the lesser curvature twenty-five times and six times on the pylorus. However, as revealed by autopsies, the neoplasm remains confined to the lesser curvature in only about 15 per cent. of the cases.

Clinical Description.—Cancer of the stomach begins insidiously with the symptoms of chronic asthenic gastritis, usually after the thirtieth year, and often where there has been no digestive trouble, and runs its course rapidly without a stop, and is accompanied by increasing emaciation, which the most careful alimentation is unable to hold in check, under favorable circumstances, for more than a few months. The appetite is early lost, but exceptionally it remains normal. The symptoms are at first digestive, but encroach more and more on the period when the stomach should normally be at rest, and consist of a sensation of heaviness or weight, to which are soon usually added pain, nausea, and vomiting. Indeed, two clinical groups of carcinoma may be distinguished. In the one group pain predominates, and is accompanied by periodical vomiting. In the other group there are only mild symptoms, such as digestive discomfort, heaviness, flatulency, and

eructations or belching. The commencement is often sudden, and marks the end of a long period of good digestion. The course may be characterized by remissions and short periods of improvement—little breaks in a cloud that never lifts; but death usually occurs, from self-poisoning and starvation, in from twelve to fifteen months after the first clinical manifestations. In some cases there may be no gastric symptoms for a long time, the disease being manifested only by progressive emaciation and weakness. But these latent cases are infrequent. The clinical picture varies accordingly as the cancer involves the cardia, the body of the stomach only, or the pylorus.

Carcinoma beginning on and limited to the cardiac orifice is very infrequent, the entrance to the stomach being in nearly every case involved secondarily by extension of the neoplasm from the adjacent region of the esophagus or stomach. The trouble begins with a feeling of fullness beneath the tip of the sternum, usually first noticed during eating or soon after a bolus of solid food is swallowed. A swallow or two of fluid relieves the sensation, which returns from meal to meal, and daily forces itself more and more upon the attention. The appetite begins to fail, and the feeling of fullness and weight becomes more obstinate. The patient soon learns that fluids pass into the stomach more readily than solids, which seem to stick behind the sternum. There are scarcely ever sharp, prolonged attacks of lancinating pain; indeed, there is seldom any severe pain, but merely at times a little dull, burning sensation, associated with the consciousness of the presence of a foreign body which can not enter the stomach. Neither swallowing nor external pressure with the hand perceptibly increases or relieves the peculiar discomfort. Emaciation, which began with the first symptom, increases more rapidly as the food is restricted, solids being excluded as a result of observation, and the poor appetite further cuts off the quantity of food. But the emaciation is due not only to the increasing cardiac obstruction and to the self-imposed fasting, but also to the increased nitrogenous waste attributable to the cancer itself. As the obstruction increases food collects above it and dilates the esophagus. The fermentation and irritation cause some burning pain and esophagitis. Shooting pains, somewhat severe in character, which have no connection with the meals, are sometimes experienced. The food, mixed with mucus, is expressed or regurgitated into the mouth, and chemical analysis shows that it has not entered the stomach.

If the cancer be of the soft variety, a marked diminution of the obstruction may occur by ulceration, and false hopes may be excited. The inanition and the cancerous intoxication, aided sometimes by slow hemorrhages, bring life to an end in from six to ten months after the first feeling of heaviness was experienced.

Carcinoma of the body of the stomach begins with loss of appetite, with digestive trouble, consisting chiefly of a sensation of heaviness after meals, or with loss of strength and weight out of proportion to the mild local gastric disturbance. There is often a little belching of gas or regurgitation of a sour and bitter fluid. Fats and meats become particular objects of disgust. The signs of fermentation become more pronounced, and pain is added to the heaviness which encroaches more and more on the period of normal rest of the stomach. The pain is usually dull, little influenced by the taking of food or by vomiting, occurs during the period of normal gastric rest, and may persist night and day. The pain as the disease advances may change in character and become at times lancinating, and the suffering may be horrible. The food is often vomited, without producing nausea, as a rule, but sometimes with a good deal of retching. The vomit may also consist of mucus and saliva, and is likely, particularly if there is extensive ulceration, to contain altered blood and to be a dirty chocolate-brown or like coffee grounds. The patient is now pale, haggard, straw-colored, cachectic. Running throughout the evolution of the disease and preserving its clinical continuity are the progressive loss of strength and the emaciation. After twelve to fifteen, or, rarely, eighteen, months the patient dies, a helpless skeleton. Cancer of the body of the stomach may be latent, and throughout the course of the disease there may be no gastric or digestive symptom to create a suspicion of the location and nature of the trouble. The only sign, apart from those furnished by the functional and bacteriological examinations, is the progressive and uncontrollable emaciation.

Cancer, in contrast with the disease involving the cardia, more frequently affects primarily the pylorus. Primary cancer of the pylorus rarely extends to the duodenum, but may spread to the body of the stomach. Frequently the pylorus is also secondarily involved by the growth of the neoplasm, beginning on the body of the stomach, particularly the lesser curvature. The clinical expression is modified by

the affection of the orifice, and the signs of stagnation or retention appear early and predominate. In cancer of the body of the stomach there is stagnation and retention, but there are no signs of pyloric obstruction. Where the disease involves the pylorus, vomiting is more frequent and copious, fermentation more active and mixed, starvation more rapid, and to the ordinary loss of body fat and the excessive nitrogenous waste and loss of strength are added the pernicious effects of insufficient water to supply the needs of the body. Thirst is often annoying.

Symptoms.—The clinical commencement of carcinoma is commonly sudden, though the symptoms in the beginning do not often seem to be serious. The middle-aged patient sometimes boasts of the good stomach which he has long and uninterruptedly enjoyed, and is at a loss to explain the causation of his trouble. But it should not be imagined that a disease of the stomach is a protection against carcinoma. Ulcer, indeed, seems to furnish within narrow limits a favorable opportunity for the development of the disease. Traumatism, dietetic excess, or some trivial cause, occasionally marks the beginning of the clinical period.

The appetite is almost invariably poor. It is better preserved when the cardia is affected than when the cancer involves the body of the stomach or the pylorus, and sometimes remains about normal, particularly in carcinomatous ulcer and in cancer in the early period of life. The more active and varied the gastric fermentation and intestinal putrefaction, the more pronounced is the anorexia. There is often a strong disgust for meats and fats, and this may be an early symptom. It seems that the dislike for fats is greatest when butyric acid fermentation exists. The disgust for meats is absolute when the contents of the stomach have a putrefactive odor, and the instinctive exclusion of meats is in keeping with the loss of power to digest them. The anorexia —which, as a rule, tends to become complete—is voluntarily resisted by the patient, and is in vivid contrast with the evident needs of nutrition.

A sensation of heaviness and fullness is an early symptom of carcinoma which is almost never absent, but is in no respect characteristic of the disease. Like that of chronic gastritis and of myasthenia, it appears soon after taking food. The sensation usually persists as long as food remains in the stomach, and, consequently, increases with the development of stagnation and retention. In cancer of the cardia its location is beneath the lower end of the sternum, but sometimes

it is referred lower down. When the neoplasm is situated on the body of the stomach or involves the pylorus, the sensation of weight and fullness may extend over the whole area of the stomach, and is often most distinct at the lowest point of the greater curvature.

Cancer is preëminently a painful disease, but a few cases run their entire course without pain. These exceptional painless forms are more frequent in old age. The pain of carcinoma is not characteristic, but still has many distinctive features. It is sometimes strictly localized, and this occurs most frequently when the pain is not very intense; but more often it is diffuse, dull, coming in exacerbations, or lancinating, tearing, and radiating into the back or beneath the sternum. It does not depend for its existence on the taking of food, but may be increased by irritating food or by food which becomes so after its sojourn in the stomach. It is not perceptibly, or only temporarily, relieved by vomiting, and it does not cease when the normal period of gastric digestion is passed. It is often a more or less continuous pain, with exacerbations occurring independently of digestion during the day and during the night.

There is no very close and constant relation between the location of the spontaneous pain and the situation of the neoplasm; but in cancer of the cardia a dull, aching, and sometimes shooting pain is frequent in and about the left shoulder-blade. The coincidence of an interscapular pain with the location of the neoplasm on the lesser curvature has often been noticed. The pain may be in the loins when the neoplasm is located on the posterior wall. Epigastric and dorsal points sometimes exist when the cancer ulcerates. When the cancer involves the pylorus, the pain due to the malignant growth may be located in the epigastrium or the left or right hypochondrium, but the pain due to obstruction is often characteristic. The pain, then, often appears during the period of digestion in paroxysms, colicky in character, and coinciding often with gastric peristalsis, visible on the thin abdominal wall. This pain of obstruction is completely relieved by emptying the stomach or by gastro-enterostomy.

Vomiting is not an early symptom of cancer of the stomach, but occurs irregularly, and usually three or four months after the beginning of the clinical period. When the neoplasm involves the cardia, the fluids may enter the stomach, and the little regurgitant vomiting occurs only during or immediately after a meal of solid food. But at a more advanced period, when the esophagus is dilated above

the obstruction, the regurgitation may recur much later, even during the period of rest of the normal stomach. The food is for a time retained in the esophageal sac. As the obstruction becomes more complete the regurgitations may consist simply of swallowed saliva and mucus. Before the fatal termination the obstruction is sometimes removed by ulceration, and the regurgitation ceases; but this seldom occurs.

A few cases of cancer of the body of the stomach, particularly when well, treated, run their course without vomiting. Vomiting is likely to be infrequent when the neoplasm is located on the posterior wall, or when it is of the hard variety and infiltrates the whole body of the stomach. It is, however, often uncontrollable, and produces great distress when the neoplasm is located on the lesser curvature. The vomiting is most frequent after taking food and during the period of digestion, but it may occur when the stomach should be empty, and may consist of the mucus and saliva swallowed, or particles of retained food may be brought up with much retching.

Vomiting always occurs when the pylorus is involved, and is often profuse on account of the obstructive retention. In pyloric cancer the vomiting is most common two or three hours after a meal, but may occur at any moment.

Hematemesis is observed clinically in about 40 per cent. (Brinton) of the cases of carcinoma of the stomach. As a rule, the hemorrhages are small and the blood passes with the gastric contents into the intestines, or it appears in the vomit after having undergone partial digestion or putrefaction in the stomach. The vomit is then more or less colored by it, and is often like coffee grounds. The coffee-ground vomit is in no manner, when taken alone, characteristic of carcinoma, but is met with in ulcer, passive congestion, erosions, ulceration, chronic gastritis, and varicose esophageal veins. The hemorrhage of cancer is seldom sufficient to excite vomiting, but sometimes a large vessel (pyloric artery) is opened and a profuse and fatal hematemesis results.

Signs.—The **physical signs** of cancer of the cardia are very important, but not always distinctive. Percussion and pressure over the lower end of the sternum are often painful, and sometimes exquisitely so. No tumor can be felt or seen. The second deglutition sounds are sometimes absent, but they are more often delayed. Rarely, the tumor, by pressure on the aorta, produces a systolic murmur, and makes it difficult to exclude the existence of an aneurysm, which should always be

done before the tube is used. An attempt should first be made to pass a large stomach-tube, and if this passes readily into the stomach, the trouble with swallowing which has prompted the examination is most likely due to spasm of the cardia. But this is not necessarily the case, as even a large sound sometimes passes readily in the early stage of carcinoma or where the cardiac orifice is made free by ulceration of the neoplasm. If such be the case, a little blood and mucus will probably be found in the eye of the tube, and sometimes nests of cancer cells or pieces of the neoplasm. If the stomach-tube is arrested, and no contents of the dilated pouch can be aspirated, a little water may be allowed to flow in, while a gentle effort is made to push the tube further on. In case of failure, the fluid should be aspirated and saved for examination, and the tube withdrawn, and the distance to the obstruction from the incisor teeth should be measured. An effort may be made to pass soft and smaller esophageal sounds, and in case of failure, and no evidence of the nature of the obstruction having been obtained, the examination may be repeated after the administration of a dram of bromid of potash in two or three doses. If the obstruction be again met with, and it is situated about 40 cm. from the incisor teeth, if the patient is beyond thirty and the difficulty has been steadily growing since its commencement a few months previous with loss of appetite and emaciation, there is then not much doubt that there is a malignant growth involving the cardiac orifice of the stomach. The stomach itself is abnormally small and retracted, and the intestines are also likely to be empty. The digestive tube becomes more and more contracted and empty as the obstruction increases, and the thin abdominal wall recedes as the abdominal contents decrease.

In every case of cancer of the stomach a tumor exists. Early in the disease, at the primary site, a neoplastic mass forms; and later, secondary deposits develop. But the primary tumor can not always be detected. It may be located on a part inaccessible to physical examination, but the displacements of the stomach which so frequently exist sometimes remove this difficulty. The primary growth may be covered by an enlarged liver or concealed by ascites. The fullness or emptiness of the stomach may reveal or conceal the tumor. In the search for the tumor it is highly important to proceed systematically. The examination should be made when both the stomach and bowels are empty. Ascitic fluid should be withdrawn. The examination is not complete until it has also been made while the stomach is dis-

tended with air or gas. The primary growth will in this way be more likely to be detected and properly located. If this plan be adopted, in at least four-fifths of the cases of cancer of the body of the stomach and of the pylorus a tumor will be detected at some time during their evolution.

A palpable tumor is not an early sign of cancer of the stomach, and is not often detected before the beginning of the last six or eight months, but sometimes earlier and sometimes nearer the end. At first may be noticed a circumscribed, resistant area, which is seldom sensitive. The tumor grows and changes its character, becoming knotty, irregular in consistency, larger, adherent, sometimes exquisitely tender, but more frequently manifesting only a little more pain on pressure than do the surrounding parts. The neoplasm often feels harder and larger on palpation than it is in reality after allowance is made for the surrounding inflammatory swelling. Even the soft infiltrating cancer produces a palpable tumor.

The physical signs of cancer of the body of the stomach may be of the greatest diagnostic value. These signs consist almost exclusively of the physical evidences of a tumor possessing the particular characters of a malignant growth. Cancer of the posterior wall and of the portions of the anterior surface and the greater curvature which are covered by the left ribs often can not be detected by physical examination. Situated on other parts of the body of the stomach, the characteristic tumor may often be found; or it is first found when these parts become involved by extension.

Cancer of the lesser curvature may lie beneath the ribs and the left lobe of the liver, and be inaccessible. But in the large majority of cases, during the second half of the clinical period, it can be detected if the stomach be empty, and much more readily if the stomach, as often happens, is displaced downward or vertically. On inspection, the tumor, well defined above and below, may be seen moving up and down on inspiration and expiration, emerging from beneath the costal arch toward the end of inspiration and disappearing from view as the diaphragm rises. On palpation, the tumor, visible or invisible, can often be felt, and also fixed on expiration, unless it be firmly adherent to the diaphragm. This relation of the tumor to the movements of the diaphragm is a very important, and, when taken in connection with its location beneath or just below the costal arch, and with the position of the body of the stomach below it, is an almost characteristic sign of a tumor of the lesser cur-

vature of the stomach. If the fingers be laid flat and gently on the abdominal wall, the tumor glides up and down with expiration and inspiration respectively. If at the end of inspiration the tips of the fingers be gently but firmly pressed above the tumor, the mass can be arrested, and when released near the completion of expiration it slips up from beneath the fingers. Another important characteristic of a tumor of the lesser curvature is its inaccessibility when the stomach is full. It can not be pushed to either side.

If the cancer is located on the portion of the greater curvature uncovered by the ribs, the tumor is situated near or beneath the umbilicus, moves with respiration, is easily fixable on expiration, and is most accessible when the stomach is full. The tumor is sensitive, commonly knotty, but is sometimes smooth, and usually possesses a border irregular in consistency but at points very hard and sharply limited. When not adherent it is pretty freely movable up and down, and to a limited extent to either side.

Cancer of the accessible part of the anterior wall is most easily felt when the stomach is moderately distended with gas, the artificial distention with air or an effervescent powder being far preferable to distention with food. The tumor is more or less sensitive, knotty, and here and there the irregular border is hard. If the cancer is an infiltrating scirrhus, the stomach is smooth, small, and resistant. The wall is non-elastic, and inflation with air or gas produces severe pain without increasing the size of the stomach. If the tumor is situated on the posterior surface, it may be felt when the stomach is empty, but it disappears when the stomach is full or distended. Clinically, what is sometimes felt during life is not in reality the primary growth, but secondary deposits in the omentum or in the left lobe of the liver.

In cancer of the body of the stomach the organ is normal in size or smaller than the average. If it be enlarged, the increase in size is an accidental association. A tumor of the body of the stomach sometimes pulsates on account of being in contact with the abdominal aorta. The pulsation is lifting, and not expansive as in aneurysm. If the tumor be free, the pulsation ceases in the knee-chest position, and when the mass is pushed to either side so as not to come in contact with the aorta. But the tumor may be adherent, surround or compress the aorta, and produce a systolic blowing and whirring murmur and diminution of the arterial pulse below. The differential diagnosis between an aneurysm and a can-

cerous tumor of the stomach depends in such a case on the other signs or symptoms which are present and belong only to the one or the other disease.

The tumor of pyloric carcinoma is seen or felt to the right, on, or sometimes to the left of, the median line. It may be invisible and inaccessible to palpation, and lie deep beneath the liver. But, fortunately, in pyloric cancer the stomach is commonly displaced, and its palpation is made thereby possible and easy. The tumor moves with respiration, and unless adherent is fixable on expiration. But cases are not rare where the pyloric tumor is exceedingly movable, and may be pushed about in the right iliac fossa (where it is sometimes found) and across the median line. On inflation of the stomach the tumor of the pylorus descends to the right and downward; but it may also be displaced upward and to the right, or simply downward, in case the tumor is bound by adhesions. The stomach is not always increased in size in cancer of the pylorus, even when obstructive retention exists, and peristalsis may be strong and visible. When the cancer converts the pylorus (by ulceration or by infiltration) into a rigid and incontinent ring, the air may be heard rushing through the pylorus with each compression of the inflating bulb.

Naturally, a gastric tumor is not a pathognomonic sign of cancer, but gathers a good deal of its value from the associated symptoms and signs. It aids in the location of the disease and in the selection of the proper treatment.

The emaciation is not characteristic, and may be no greater than in other diseases. But the skin does not preserve its clear and rosy color, as in the extreme emaciation of some nervous affections; nor is all the fat lost, as in simple chronic inanition. The body albumin early and progressively disappears, and the emaciation and cachexia differ from the pale, edematous appearance of chronic nephritis. The emaciation of carcinoma resembles that produced by lack of food and water. The body is not soggy, but the skin and both other tissues are abnormally dry. But localized temporary edema is common enough in carcinoma of the stomach. It is first noticed about the ankles, and may appear early in the disease. It occurs without albuminuria or cardiac insufficiency, and comes and goes without any changes in the diseased blood. The edema is sometimes confined to one extremity, and is due to phlebitis. The occluding thrombus, favored in its formation by the diminished alkalinity of the blood, is most frequent in the veins of the lower extremities,

but may also occur in other veins. Localized edema due to venous thrombosis, or fugacious edema, unassociated with renal or cardiac insufficiency, or with diarrhea should excite suspicion of malignant disease. But, as the rule, the cachectic emaciation of carcinoma is progressive and dry.

The state of nutrition found in carcinoma of the stomach is due partly to starvation. The appetite is diminished, and nearly always, in all forms and localizations of carcinoma, too little food is eaten to supply the needs of nutrition. Temporary improvement may consequently often be obtained by the prescription of a carefully selected and sufficient diet. The emaciation in obstruction of the cardia and pylorus is also due in part to an insufficient absorption of food. It is very common for increase of weight and strength to follow when the obstructed cardiac entrance is made free by ulceration. Improvement in the state of nutrition may also be obtained by surgical operations facilitating the introduction and utilization of food, and sometimes by rectal feeding. Still another factor of the emaciation and loss of strength in cancer of the stomach is the failure to digest and to utilize the small quantity of food which is eaten. The active gastric fermentation entails a heavy loss of non-nitrogenous food, and the almost constantly large quantity of indican in the urine and the foul odor of the stools reveal the activity of putrefaction in the intestines. The continuous absorption of the products of fermentation and putrefaction exerts a deleterious influence on nutrition.

But there is still a peculiarity of catabolism present in carcinoma which starvation and fermentation and putrefaction do not explain. This is the excessive nitrogenous elimination due to the active destruction of body albumin. This excessive nitrogenous waste is uncontrollable by diet, is due to the malignant growth itself, and creates a close resemblance in this respect between carcinoma and the infectious diseases. It seems probable, both from reasoning and experimentation, that a protoplasmic poison is formed by the neoplasm and circulates in the blood, to which is due the chemotaxis, hematocytolysis, leukocytosis, and excessive destruction of the albuminous tissues of the body.

These many factors of the emaciation, cachexia, and loss of strength are not equally active in all cases of carcinoma of the stomach. Consequently, too great stress should not be laid on the state of nutrition at a particular stage in the evolution of individual cases. The body may remain pretty well nourished until late in the second half of the course of the

disease. There may be short and temporary improvement and gains, which should not be allowed to deceive. The emaciation may be gradual and progressive from the beginning, or may begin suddenly in the course of the disease and proceed rapidly. The loss of weight, the weakness, the selective nitrogenous waste, and the dry cachexia should, nevertheless, excite suspicion of a malignant disease. But these signs are not worth much in the early diagnosis of cancer of the stomach.

The **functional signs** of carcinoma of the stomach are very valuable aids to a correct and early diagnosis. Neither the hypochylia, nor the motor insufficiency, nor the diminution of absorption are pathognomonic signs, but they possess both a positive and a negative value. Normal secretion and a good motor function speak emphatically against cancer. Supersecretion and hydrochloric superacidity possess great negative value and exclude cancer where there is no history of ulcer or of hypersthenic gastritis.

In respect to the functional signs carcinoma may be divided into three forms, according to the situation of the neoplasm on the body of the stomach or at one of its orifices. In cancer involving the cardiac orifice the functional signs are variable; there may be normal secretion and normal motor activity throughout the disease, except in so far as the functions are modified by subnutrition and anemia; or secretion may be diminished if secondary asthenic or atrophic gastritis should develop. In cancer of the cardia the functional gastric signs are not required for determining the location of the disease, and they give very unimportant information concerning the nature of the disease. In regard to the motor function, it makes a great difference whether the neoplasm is confined to the body of the stomach or whether it obstructs the pylorus. In the description of the secretory signs the situation of the cancer on the body of the stomach or close to the pylorus need not be kept in mind; for the secretory changes are due to the nature and extent of the accompanying gastritis, and, to a less degree, are the effect of the subnutrition, of the toxemia, and of the anemia.

Secretion in carcinoma is diminished early in the disease, often before a tumor can be detected; and the diminution is persistent from day to day, and often rapidly progressive. In a small number of the cases of cancer nutrition is fairly well maintained, and the associated gastritis is slight, and secretion can remain moderately active until near the fatal termination. But the rule, nevertheless, stands that in carcinoma of the

stomach the free hydrochloric acid soon disappears, and gradually the combined hydrochloric acid decreases in the contents obtained after a test-breakfast. Simultaneously, the pepsin and labferment and their mother-substances likewise decrease, until eventually the albuminous foods undergo no hydrochloric-pepsin digestion. The ferments, as in asthenic gastritis, diminish in proportion with the diminution of hydrochloric acid (H + C) secretion. The hypochylia is not due to the malignant growth itself, though the alkaline transudate from the blood-vessels and the cancer juice doubtless often neutralize some of the hydrochloric acid secreted. Nor is it due to the state of nutrition and to the blood alone, though the progressive cachexia may be an active factor. The same secretory changes occur when the cancer affects another organ, as the uterus or the breast. But the state of secretion is due chiefly to the associated asthenic gastritis. The secretory signs are only indirect manifestations of the cancer, and are in themselves in no wise pathognomonic. Gastric absorption is diminished, and the diminution is persistent and progressive.

When the cancer is engrafted in ulcer it may be accompanied by secretion as rich as normal until near its fatal termination; but it is more common for secretion to diminish as the disease progresses beyond a certain period. In one of our 38 cases the cancer developed during the course of an old hypersthenic gastritis (no ulcer), and hyperchylia persisted to fifteen days before death. In about ten per cent. of the cases of carcinoma secretion remains nearly normal in acid and in ferments until near the end of the disease. In nearly 20 per cent. of the cases hypochylia develops slowly. But in the remainder of the cases (about 60 per cent.), hypochylia begins early and develops rapidly, free HCl disappearing, and the digestive power of the contents varying from 20 to near 0, when estimated by the method of Hammerschlag.

The **motor insufficiency** is a more direct result of the malignant growth. The muscular layer becomes infiltrated with cancerous cells and edematous from obstruction of the lymphatic circulation. Late in the disease the whole muscular system becomes weak from toxemia and wasting. The neoplasm often more or less involves and obstructs the pylorus. The motor insufficiency is, in a large percentage of the cases, an early sign, but it becomes pronounced more rapidly in pyloric carcinoma. For a variable period stagnation exists; first stagnation of solids only, and later of

both solids and liquids. Retention follows, and the stomach then always contains food and liquid. Retention occurs early and almost invariably in pyloric carcinoma. The only two exceptions which we have seen were cases of scirrhus—the pylorus being converted into a functionless ring, the stomach being small and evacuating its contents with abnormal rapidity, achylia being complete, and lactic acid persistently absent. In cancer of the body of the stomach stagnation is an early sign in about 60 per cent. of the cases; in a part of the remainder stagnation appears late in the disease, and about five per cent. of the cases run their course without motor insufficiency. Most important characteristics of the motor insufficiency of cancer are its persistence and its frequently rapid increase.

The **bacteriological signs** are in certain circumstances characteristic of cancer of the stomach, and may be so even at an early stage, when no tumor can be detected. In no other disease is fermentation more active, for no other disease furnishes so many favorable conditions—stagnation, retention, hypochylia, diminished absorption, and prolonged salivary digestion.

The form of fermentation is not always the same. Before the disappearanee of the free hydrochloric acid and the beginning of motor insufficiency, there is no fermentation except what may occur accidentally and temporarily. As soon as stagnation begins, and before the free HCl disappears, the fermentation is sometimes due to yeast, but is most frequently butyric. In carcinoma of the stomach butyric fermentation is almost as common as lactic acid formation, but it is neither persistent nor characteristic. As the stagnation and hypochylia increase, the fermentation becomes bacillary and chiefly lactic. In no other disease of the stomach is lactic acid so frequently formed in large quantity, when certain precautions are taken to prevent its introduction or its retention in the stomach from a previous meal. In the thoroughly washed stomach lactic acid is formed from food which is perfectly free from it, in quantity greater than one per thousand, in two-thirds of the cases of carcinoma of the stomach.

The formation of lactic acid takes place in the human stomach only in very special conditions, and these conditions are most frequently present in carcinoma. In the first place, it is absolutely essential that hypochylia exist to such an extent that no free HCl is present in the contents obtained one hour after the test-breakfast. Even the presence of combined HCl in moderate quantity suffices to arrest the formation of

lactic acid, unless motor insufficiency is so great as to cause retention. Consequently, lactic acid formation is more frequently and persistently associated with absent free HCl and nearly normal combined HCl in malignant than in benign obstruction, unless the cancer develops on an old ulcer or in the course of hypersthenic gastritis; for the hypochylia of carcinoma is persistent and progressive, all the constituents of secretion being involved. Lactic acid formation occurs in cancer at a period when the motor insufficiency is not so great as is required for lactic acid formation in benign diseases. This clinical fact is probably due to the accumulation of lactic acid forming bacilli over the portion of the gastric wall rendered stiff and motionless by the neoplasm, and to the existence of food retention in a smaller quantity, but persistently, in carcinoma, at a period when the general motor insufficiency is comparatively not great. Lactic acid may be formed in carcinoma if the tumor is extensive (on body) and uneven, at a time when the stomach evacuates its contents in a nearly normal period; but lactic acid formation never occurs, under such circumstances, in a benign disease. The conditions of lactic acid formation are hypochylia with no free HCl, motor insufficiency, and the accumulation of vigorous lactic acid producing germs. These conditions are seldom persistently and progressively fulfilled to such a degree that more than 1 per 1000 of lactic acid is formed during the digestion of the test-breakfast on the morning following thorough lavage on the preceding evening, unless the disease of the stomach is cancer.

Lactic acid formation occurs invariably in cancer of the pylorus when the obstruction becomes sufficient to produce retention and there is no free HCl in the contents after the test-breakfast. But lactic acid formation may occur in benign retention with hypochylia, and great precaution must be taken in interpreting lactic acid formation in obstructive retention. In two-thirds of the cases of cancer situated on the body of the stomach there is lactic acid formation in quantity greater than 1 per 1000. Consequently, the absence of lactic acid formation does not exclude cancer of the pylorus before retention occurs; nor does it exclude a cancer of the body of the stomach, whatever be the stage of its growth. In 109 cases of disease of the stomach with hypochylia we have found lactic acid in quantity greater than 1 per 2000 in 44 cases; 38 of the 109 cases were carcinoma, and in 31 of the 38 cases lactic acid formation was present. In 132 cases with hyperchylia we have found cancer 3 times—2 engrafted on ulcer and 1 on

chronic hypersthenic gastritis. Hammerschlag, in 250 cases of diseases of the stomach, found lactic acid in 35, and 29 of these 35 cases were cases of carcinoma. In 153 cases Strauss found lactic acid in 27 and cancer in 22. In 14 cases of cancer of the stomach Klemperer found lactic acid in 12; in 42 cases of the same disease Hammerschlag reports no free HCl in 37, digestive power less than 20 per cent. in 30, and lactic acid in 26; Boas found lactic acid in 30 out of 40 cases. In 55 cases of cancer of the stomach Hayem found achylia in 6, hypochylia with no free HCl in 48, hyperchylia (with disappearance of free HCl in one month) in 1, and lactic acid in 25. These statistics give a general idea of the frequency and diagnostic value of lactic acid formation in cancer of the stomach.

When, in the course of cancer, obstructive retention develops, the fermentation becomes mixed. The gas-forming germs now become active. The kinds of micro-organisms change during the course of cancer, as do the forms of fermentation. Sarcinæ are only found when retention coexists with secretion so active as to leave HCl free; lactic acid fermentation destroys them rapidly, and they are not found in association with it except isolated and dying. Lactic acid formation is accompanied by the bacillus geniculatus. In the mixed fermentations are found bacilli and yeasts. There is no H_2S formation in malignant disease of the stomach (Boas).

The **anatomical signs** are inconstant, but may be absolutely demonstrative, revealing the malignant disease to the eye. The washings and vomit should be repeatedly and persistently examined for pieces of the tumor. A negative result is without meaning, but the discovery of little pieces of the mucous membrane showing the histological characters of asthenic gastritis does not exclude carcinoma.

Another very important anatomical sign is the presence of pus and cancer cells in the vomit or expressed contents. There is no pepsin-hydrochloric digestion, or very little, in the stage of carcinoma when ulceration is active. Consequently, the dead tissue and the inflammatory products are not digested as in ulcer, and the motor insufficiency of carcinoma makes the obtaining of them in the vomit and washings all the more likely. It should not be forgotten that the pus may be swallowed, and it is only a confirmatory sign, except when mixed with the debris of cancerous tissue which can be recognized as gastric.

The **blood changes** in cancer of the stomach are not in themselves characteristic, but are very marked, progressive, and possess some diagnostic value. The anemia may be mild,

severe, or grave, and is not accompanied, as a rule, by hemic murmurs in the heart and the blood-vessels.

Cancer of the stomach produces greater changes of the blood than does cancer of any other organ of the body. The pathological blood alterations are due to subnutrition, to hemorrhage, to toxemia, and to the low reparative power of the body, and affect the development of the blood, the vitality and resisting power of the corpuscles, and the composition of the plasma.

Coagulation and fibrin formation are normal or less active than in health. Rapid and excessive fibrin formation does not occur in cancer unless it is complicated by inflammation.

The specific gravity of the blood is always reduced, and to a greater extent than would be indicated by the diminution of the hemoglobin, unless leukocytosis and other counteracting influences are present. The specific gravity of the blood, which can be easily estimated by the method of Hammerschlag, is determined by the number and size of the red corpuscles, by the quantity of hemoglobin, by the quantity of water, by the richness of the plasma in albumin and salts, and by the number of leukocytes. In cancer there is always a diminution of the hemoglobin below the percentage which would be indicated by the number of red corpuscles; but the average size of the red corpuscles may be diminished, the quantity of water may be so greatly reduced as to produce oligemia sicca, the plasma may be poor and thin, and the number of leukocytes may be greatly increased. Consequently, the specific gravity does not increase and decrease with the percentage of hemoglobin; but it may so happen that the combined effect of the other factors is multiplied, or they may work more or less in unison to reduce or increase the specific gravity. The specific gravity of the blood is characteristic of the blood of cancer only when its relation to all the factors which influence it is known.

In cancer of the stomach the quantity of hemoglobin and the number of red corpuscles steadily decrease from the moment the blood is altered by the malignant disease until death arrests the process. There is in reality progressive descent; but this descent may be masked by the occurrence of oligemia sicca, which may cause the percentage of hemoglobin and the number of corpuscles to approach or return to the normal percentage and number. But the oligemia sicca only masks the anemia, which is easily detected by the microscope and by stains.

The hemoglobin percentage in cancer of the stomach is

always less than the percentage of red corpuscles, because the average size of the red corpuscles is nearly always less than the average normal size, and the regeneration of hemoglobin is deficient. The persistence of this condition in the absence of signs of regeneration of the blood is a marked characteristic of carcinomatous blood.

The blood's losses in cancer of the stomach are nearly always permanent. Even after an operation, when blood is destroyed by the anesthetic and lost by hemorrhage, the loss is very slowly and incompletely restored, even though the operation greatly improves nutrition and the functions of the stomach. The cancer produces obstinate insufficiency of the blood-building organs.

The red corpuscles undergo qualitative alterations. The average size is less than normal, and a glance at a specimen under the microscope will reveal the large number of microcytes, rendering it seldom necessary to compare the volume percentage given by the hematocrit with the percentage of counted red corpuscles. But in the grave stage of the anemia megalocytes may appear; but many of these are imbibition corpuscles. The red corpuscles also degenerate and undergo destruction in the circulating blood. The blood becomes poor in chlorids and nitrogenous substances, and a protoplasmic poison is formed in cancer of the stomach.

The white corpuscles in cancer of the stomach may undergo quantitative and qualitative alterations. The number of white corpuscles is decreased by the subnutrition and by the low vitality; and their number is increased by hemorrhage, by hematocytolysis, by metastases, by peritonitis, and other inflammatory complications, and by strong reaction of the organism when it still possesses some power of resistance. The number of white corpuscles is the result of the struggle between these factors, and leukocytosis is present in more than half of the cases. But cancer of the stomach, particularly when it obstructs one of the orifices, and when it is not accompanied by an inflammatory complication, may run its entire course with a normal or diminished (terminal leukopenia) number of leukocytes. During the clinical stage of carcinoma of the stomach digestive leukocytosis seldom occurs. The insufficient blood-building organs do not respond to the demands of digestion, absorption, and assimilation. The number of white corpuscles three hours after a rich meal, including proteids and fats, is not materially different from the number of leukocytes counted before the meal. Myelocytes are frequently found in the blood of cancer, and the leuko-

cytes undergo degeneration in greater and greater numbers as the blood alterations increase. The nucleus of the lymphocytes becomes poorer and poorer in chromatin as the cytoplasm disappears; loses its form and structure; enlarges and finally undergoes dissolution. The polynuclear leukocytes are affected by hyperchromatosic degeneration. As many as ten per cent. of the leukocytes may be degenerated, and these degenerate corpuscles go to pieces in the circulation, appearing as formless protoplasmic masses in the fresh and the stained preparations of the blood.

The alkalinity of the blood is diminished in carcinoma of the stomach. This is doubtless due, in part, to the increased destruction of body albumin, whereby sulphuric, phosphoric, lactic, and oxybutyric acids are set free. But the excessive acid fermentation and the putrefaction of which the digestive tube is the theater also exert a noteworthy influence.

The **urine changes** in carcinoma of the stomach are very marked, but no one singly, nor all taken together, enable us to infer the existence of the cancer. The normal digestive curve of diminishing acidity, which reaches its highest point between three and five hours after a meal, is seldom found in cancer of the stomach, and this is plausibly explained by the small quantity of hydrochloric acid secreted. The twenty-four hours' urine may be constantly almost neutral, and this may be due to the absorbed organic acids and ammonia produced by gastro-intestinal fermentation and putrefaction. On the other hand, the acidity of the urine is often increased, and uric acid precipitation is frequent. The reaction, though presenting variations, is of no diagnostic value.

The uric acid elimination is sometimes largely increased, as would naturally be expected, in leukocytosis, in active destruction of body albumin, and in acid toxemia. The secondary insufficiency of the liver seems also to exert some influence in causing uric acid precipitation. Almost constantly, in cancer of the stomach, more nitrogenous matter is eliminated than is introduced with the food. The excessive nitrogenous elimination represents the excessive destruction of body albumin; but on account of the insufficiency of the food the total elimination of nitrogenous matter is commonly less than in health. When little food is digested and utilized and the emaciation is advanced, the elimination of urea, and also of the chlorids, falls very low.

The urine often contains urobilin, particularly when the liver becomes secondarily involved or when the red cells rapidly disintegrate. Indican is largely and almost con-

stantly increased. Albumoses may also be found in the urine, particularly when the neoplasm undergoes rapid ulceration. Acetonuria is a frequent terminal sign, and the urine may contain, particularly where there is coma, traces of oxybutyric acid. The urine signs demonstrate how severely nutrition is affected, but possess, on account of not being peculiar to cancer of the stomach, an exceedingly small diagnostic value.

In the majority of the cases of cancer of the stomach the **bowels** are constipated until the beginning of the last few weeks, when diarrhea commences. Sometimes the stools are frequent, and sometimes dysenteric; but more commonly every one or two days there is a large, foul, and loose movement. In about one-third of the cases constipation persists from the beginning to the termination, an incomplete evacuation of the bowels occurring every four or five days, and the stool consisting of hard lumps of intestinal secretion, and of a quantity of undigested muscular fibers. The constipation is most obstinate when the cardia is obstructed. In pyloric cancer the course is broken by diarrheal attacks. Unmixed lactic acid fermentation favors constipation.

The stools sometimes contain blood. Traces of blood are frequently found, and melena is usual when the vomit contains much blood or is colored like coffee grounds.

Carcinoma of the stomach is an afebrile disease. But **fever** may occur during its course, and may be due to a complication, such as perigastritis, subphrenic abscess, peritonitis, pleurisy, pericarditis, or to a secondary cancerous deposit which excites inflammation. Fever may also occur as a symptom of an associated disease; but febrile exacerbations may sometimes be present without any discoverable cause except the cancer itself or an ulceration of the neoplasm. The fever peculiar to cancer is intermittent, beginning with a chill, ending with free perspiration, and recurring irregularly, resembling closely a malarial chill, and is most plausibly explained as an acute auto-intoxication. In the cachectic stage the temperature is usually subnormal continuously, but it may rise intermittently to the normal point, the pulse becoming more rapid and the patient complaining of feeling hot. A slight rise of temperature during the height of digestion is quite common.

Cancer of the stomach sometimes ends in *coma*, which may be preceded by inanition delirium. The patient falls into a stupor, the breathing becomes deep and stertorous, the pulse rapid, the muscles sometimes twitch, or there may be

general convulsions. The coma ends fatally in from one to three days.

Diagnosis.—The diagnosis of carcinoma of the stomach may be easy, difficult, or impossible. Consequently, the physician may be certain, doubtful, or without suspicion of the existence of a cancer in a particular case. Modern methods of examination reveal the disease when formerly it would have been overlooked.

From a diagnostic view-point the cases can be divided into two large classes, accordingly as a tumor can or can not be detected.

If a tumor be found, the examination should be so conducted as to determine whether the tumor is gastric and malignant, and where it is located on the stomach. When no tumor can be detected, the diagnosis may have to be made early in the disease, while radical surgical treatment is practicable; or late, when palliation is the only aim. But it should not be forgotten that a tumor so located as to be easily accessible to physical examination may be discovered early in the disease.

A tumor of the stomach may be found in almost any part of the abdomen to which the displaced or enlarged stomach extends. Consequently, the search should not be limited to the small area of the normal stomach, nor should those tumors found within this area be supposed to belong necessarily to this organ. Outside of this narrow field gastric tumors are also found. It is often necessary to differentiate the tumors of the stomach from possible tumors of other abdominal organs, such as the liver, spleen, gall-bladder, duodenum, colon, kidneys, mesentery; and also to differentiate them from abdominal abscess and from abdominal aneurysm.

Very valuable information is often furnished by the clinical history. A malignant palpable tumor of the stomach invariably produces gastric symptoms and signs. Carcinoma of other organs may disturb their functions in a manner noticeable by the patient. But the subjective symptoms should simply draw the attention in a particular direction, for they may be due to secondary deposits, to complications, or to associated non-malignant disease. The stomach is more likely to be disturbed than is any other organ, being a center for the expression of so many abdominal diseases. Consequently, the total absence of gastric disorder is a very valuable negative sign.

The physical signs are often more direct and conclusive. The tumor of the stomach can be seen or felt only in the re-

gion occupied by the stomach in the particular case. Consequently, the exact location of the stomach by percussion, by inflation, by splashing, by filling the organ with water, by palpation of the balloon sound, and by electric illumination may be of great value. Not only is the tumor thus located within or without the gastric area, but other valuable information may be obtained. After inflation the tumor may be seen or felt to belong to the stomach, and to be more clearly and widely separated from surrounding organs. The tumor, which, during inflation, was surrounded by a tympanitic area, may, after filling the stomach with water, be surrounded by a duller or flat zone, and be within the area of gastric splashing. The tumor may present a dark spot on the illuminated stomach, a phenomenon which is very interesting, but possesses very little diagnostic value unless clearer and more conclusive methods of examination are neglected. The tumor may be revealed by inflation, or may thereby be made more perceptible, or its position may be changed, or it may be palpable and visible only when the stomach is empty. The tumors of the colon and mesentery are simply pressed downward by inflation of the stomach; those of the left lobe of the liver are pressed upward and forward; and those of the spleen, downward, and outward, and forward, against the abdominal wall. The tumors of the greater curvature and the anterior wall are rolled and pressed forward; those of the lesser curvature disappear upward and backward; and those of the pylorus are pressed downward and to the right, or to the right and upward, and may be made inaccessible to palpation if the pyloric pouch is much dilated. All these special signs should be noted and taken in evidence when the conclusion is formed.

The tumors of the stomach also present peculiarities in relation to the movements of the diaphragm during respiration. The closer and more direct the connection of the tumor of the stomach to the diaphragm, the greater and more constant are its respiratory movements. It is easy to demonstrate by the gliding method of palpation that even the greater curvature of the stomach moves up and down with the relaxation and contraction of the diaphragm; and, consequently, the contention that tumors located on this part of the stomach are unchanged in their position by respiration is false. The extent of their mobility is limited by the increase of intra-abdominal pressure. The up-and-down movement is greatest when the lateral expansibility of the abdomen is small, and the intra-abdominal pressure in the part of the abdomen inferior to the tumor is least. Wherever the abdominal

tumor be located, its respiratory mobility will depend on the solidity of the pressure exerted, directly or indirectly, by the diaphragm; on the freedom of the organ from restricting ligaments and adhesions; and on the degree of resistance offered by the parts below the tumor.

It can easily be inferred that the tumors of the pancreas and kidneys, if firmly and closely attached to these organs, remain stationary during respiration; those of the liver and spleen (except when these organs are displaced and their attachments are weak) follow closely the movements of the diaphragm; and the tumors of the colon, mesentery, and a large portion of the stomach are not necessarily so greatly changed in their position by the act of respiration.

The relation of the movable tumors to expiration furnishes another differential sign. If at the end of inspiration the tumor be caught along its upper border by the examining fingers, it may or may not be capable of fixation during expiration. The tumors of the liver and spleen, unless these organs be pathologically movable, can not be thus fixed. In order to arrest the expiratory movement of the tumors of the lesser curvature and pylorus when their attachments are not weakened, a good deal of force is required; but the tumors of the colon, greater curvature, and greater omentum are more easily fixed, and when released slip up with little force. These respiratory peculiarities of abdominal tumors are converted by educated and experienced palpation into valuable differential signs.

Another palpation sign is valuable in the diagnosis of the tumors of the pylorus, which are often hard and solid to the touch. If the tumor be held beneath the fingers, particularly soon after a meal, gas can be felt bubbling intermittently through it. An intermittent pyloric spurt may often be heard with the stethoscope placed over the point. If the stomach be empty, the signs are best sought after the administration of a glass of water about the time that a meal is usually eaten, for active gastric peristalsis recurs regularly and periodically after each mealtime. When the bubbling is felt and the spurt heard under these conditions, the solid tumor can be only pyloric. The only possible source of error is an annular tumor of the intestines, which, only in very special and rare circumstances, can give rise to somewhat similar signs.

Percussion may also give some information. Percussion over a tumor of the stomach which is not strictly limited to one of the orifices is never dull if the organ contain gas. Percussion over a solid tumor situated beneath or over a hol-

low organ is tympanitic. The tumor may often be surrounded and separated from adjacent organs by a zone of a different percussion note. The note over the surrounding area may be changed by inflation of the stomach. Valuable differential physical information may often be obtained by inflating the colon with air or by filling it with water; or more information may sometimes be ascertained by simultaneously filling the stomach with gas and the colon with water; or by palpation of the introduced balloon sound inflated to about the size of the fist.

The functions of the stomach are never normal when it is the seat of a palpable new growth. Persistently excessive secretion should excite suspicion of a gastric ulcer as the cause of the tumor. A diminution of secretion may be present in the course of malignant disease, wherever located, and is hardly available as a differential sign. The bacteriological signs may not only locate the tumor in the stomach, but reveal its nature, as would also a piece of the tumor in the vomit or washings. Pus in the contents, if it has not found entrance through the cardia and is present in noteworthy quantity, signifies that an abscess has opened into the stomach or that there is ulceration and pus formation associated with insignificant gastric digestion. Pus is seldom found, but cancer cells are sometimes numerous, and both may aid in connecting the tumor with the stomach.

Tumors of the stomach must be so often distinguished from those of the liver and transverse colon that a special grouping of the differential signs is desirable. The tumors of the transverse colon produce intestinal stagnation above the obstruction or at the part of the bowel where the muscular coat is rendered inactive by the tumor. The hypertrophied wall may show visible peristaltic waves, and the fingers or the stethoscope placed upon the tumor may enable us to feel or to hear the intestinal contents bubbling through it. The tumors of the pylorus also present these signs, but in relation with visible or palpable gastric peristalsis. The tumors of the colon disturb the motor function of the bowel, but not that of the stomach. By inflation of the colon the tumors of the stomach are displaced upward, while those of the colon do not rise, but may be revealed to a greater extent, and their origin in the colon may be made clearer. By inflation of the stomach the tumors of the colon are pressed downward, those of the lesser curvature of the stomach disappear, the pyloric tumor goes downward and to the right

or is concealed by the dilated pyloric pouch, and those located on the anterior wall and greater curvature become apparently larger and less sharply defined. It should never be forgotten that the colon may lie between the stomach and the anterior abdominal wall, and before locating a tumor in this region the position of the bowel should be determined by inflation. The tumors of the colon can always be fixed on expiration.

The tumors of the liver can not be fixed on expiration unless the whole organ be prevented from rising,—which only happens when its attachments are weakened,—and they are pushed upward and forward by inflation of the stomach and colon. The tumor of the liver, however, may be secondary. Primary carcinoma of the liver, which is rare, produces rapid and great enlargement of the organ, and icterus often appears early. A tumor of the liver with round margins and a central cupping is always a secondary cancer, and if the stomach presents the functional and bacteriological signs of carcinoma, this organ is the seat of the primary disease, although no gastric tumor be felt.

A tumor of the gall-bladder is felt attached to the liver at the normal position of this receptacle. It may often, when the cystic duct is obstructed, be moved some distance to the left, toward the median line, and follows closely the phrenohepatic respiratory movements. There is no motor disturbance of the stomach unless the duodenum be compressed, when bile may flow continuously into the stomach and gastric obstructive retention may develop. The pyloric tumor may be readily differentiated by the pyloric bubbling, which may be felt with the fingers or heard with the stethoscope.

A tumor of the stomach is either benign or malignant, and it is of the utmost clinical importance that the two varieties should be sharply differentiated. Fibroma, myoma, adenoma, and gastroliths are very rare. More frequent is a palpable resistant mass formed by the whole stomach or by an inflammatory exudate.

The benign neoplasms produce no metastases, are slow in their development, produce often but slight functional disturbance, and, except when obstructing an orifice, cause no progressive emaciation and cachexia, develop often at an early age, and are neither nodulated nor hard.

The gastric tumor may be formed of the whole organ. The tumor formed by the distended stomach disappears when the organ is emptied. In only three diseases is the tumor

formed by the wall of the stomach—in chronic interstitial or fibrous gastritis, in disseminated scirrhus, and in the small retracted stomach of obstruction of the cardia. Between the benign and malignant affections the clinical history and the evolution often discriminate. Disseminated hard cancer may run a slow course, however, and metastases and enlarged glands often can not be detected. Obstruction of the cardia should excite suspicion of the small retracted stomach which does not lose its peristaltic power. In scirrhus and in cirrhosis ventriculi the stomach is an almost inert tube. Consequently, pyloric bubbling and visible or palpable peristalsis, however excited, decide against hard cancerous infiltration.

An inflammatory palpable exudate is nearly always the result of gastric ulcer. An ulcer history would decide the nature of the tumor were not cancer sometimes engrafted in an ulcer. The differentiation can not be made unless a secondary cancer deposit be found. Excessive or normal secretion, blood crises, digestive leukocytosis, simple inanition without excessive destruction of body albumin, are against the ulcer having become cancerous, which, it should also be remembered, occurs only in a very small percentage of the cases of ulcer.

The tumor is more likely to be cancerous the more closely the evolution, symptoms, and signs are those of a malignant disease. An unaccountable and insidious beginning in a person beyond the thirtieth year who has previously enjoyed good digestion, progressive evolution, increasing diminution of secretion, motor insufficiency, lactic acid fermentation produced by bacilli, emaciation, and loss of strength, excessive waste of body albumin, the inefficiency of treatment—all favor cancer.

The difficulty of determining the nature of the tumor is greater when the pylorus is involved. Is the obstruction due to ulcer, to cancer, or to benign muscular hypertrophy? If the tumor extends on to the body of the stomach, it is more likely to be cancer, and is most probably so if the bacteriological signs of cancer be present. If the tumor forms and the obstruction develops in the course of ulcer, the tumor is most likely benign, and is most certainly so if there are no functional, bacteriological, or malignant nutritive signs present.

Annular scirrhus and hypertrophy of the pylorus are so closely alike as to leave nearly always a large place for doubt. If the patient has been long under observation, the slow growth of the smooth hard tumor and the long duration of the trouble speak in favor of hypertrophy. Nutrition may

be improved materially by a proper diet and by nutrient enemata. But if the characteristic functional and bacteriological signs of cancer are not present, the trouble is most probably not malignant. Metastasis, nodulation, and enlargment of the glands close to the pylorus may characterize the annular cancer of the pylorus. The infiltrated, cancerous pyloric ring does not contract or relax perceptibly, as does often the benign hypertrophied pylorus. In both diseases pyloric bubbling may be felt, and the pyloric spurt may be heard, unless the orifice be completely obstructed.

If no tumor can be detected, one of the most valuable signs of cancer of the stomach is wanting. This may happen at any stage of the disease. The case may be presented for diagnosis before or after the development of emaciation and cachexia.

The diagnosis of carcinoma of the stomach when no tumor can be discovered, and before nutrition is so reduced as to suggest a serious and perhaps malignant disease, is a mere question of probabilities. But there are certain early signs which should not only excite suspicion, but should cause at least a careful search and a guarded opinion. And here, above all, must be considered the age of the patient, the absence of sufficient cause, and the previous good digestion of an ordinary mixed diet. If a person whose other organs are healthy, who is between forty and sixty years of age, who is guilty of no dietetic errors or excesses, without ascribable cause loses his appetite, feels a sense of heaviness or discomfort in his stomach, at first during digestion, and later when the organ should be empty and at rest, loses weight and strength in spite of good digestive hygiene and sufficient food—a suspicion of carcinoma is justifiable, and the combination of symptoms and circumstances should lead to a careful search for other signs. The form which the emaciation takes should be observed, and the excessive waste of the nitrogenous tissues (particularly both the voluntary and involuntary muscles), while the body still retains a good deal of its fat, should be carefully noted. The blood changes should also be recorded from time to time, and the number of white cells should be counted before and about three hours after a mixed meal. The secretory and motor functions should be carefully tested, and the quantity and character of the germs noted. If the functional, bacteriological, hemic, and nutritive signs become more and more like those of carcinoma, and in spite of rational treatment and a suffi-

cient diet, and if the clinical picture remains ill-defined and characteristic of no other disease, the suspicion is probably well founded. The early diagnosis of cancer can sometimes be made with a good deal of probability by close observation and study of the case during two or three weeks. The evolution of the symptoms and signs in a particular manner is somewhat characteristic, and in this respect a single examination gives little information. The functional and bacteriological signs develop more rapidly in some cancers than in others. The absence of free hydrochloric acid, the diminution of the ferments, the formation of lactic acid in noteworthy quantity (1 : 1000) in the thoroughly washed stomach after a test-meal containing no lactic acid, motor insufficiency, and the presence of lactic-acid-forming bacilli in large numbers—may in themselves, but better in combination with other secondary or confirmatory signs, establish the diagnosis.

The diagnosis of carcinoma in the absence of a palpable tumor, but after the development of the emaciation and cachexia, can often be made if the functional, bacteriological, hemic, and nutritive signs be taken in connection with the mode of beginning, with the evolution, and with the subjective symptoms. These signs and symptoms need not here be repeated, but great emphasis may be laid on their value when combined, and on their lack of meaning when taken singly and without regard to their order of development and progressive evolution. Naturally, the anatomical signs, if obtained, have the same value as when the tumor can be detected.

The location of the tumor is important both from a medical and a surgical point of view.

The tumors of the pylorus are sometimes very movable, sometimes disappear, and may be again found on reëxamination. On inflation of the stomach they move downward and to the right, or are concealed by the distended pyloric antrum. The most usual site of the tumor is on or to the right of the median line, beneath the lower border of the left lobe of the liver. It is hard and often annular, and gas can be felt bubbling through it intermittently. Pyloric cancer produces obstructive retention, enlargement of the stomach, and often visible, palpable, and active peristalsis.

Cancer of the cardia is revealed by the symptoms and the signs of obstruction obtained by the use of the sound. The stomach retracts and is constantly very small.

The tumors of the body of the stomach may be located by the physical signs when on the accessible parts of the greater curvature, anterior wall, and lesser curvature. A tumor of the posterior wall may sometimes be felt when the stomach is empty and the patient is in the knee-elbow posture; but it disappears when the stomach is inflated, and the anterior wall will be found free. A tumor of the pancreas should not be mistaken for a cancer of the posterior wall; the signs have already been given by which it can be determined that the tumor is gastric and malignant. When the tumor is located on the body of the stomach and the pylorus is left free, the weight and strength are longer maintained and treatment is of more avail.

Differential Diagnosis.—It often happens that when the patient presents himself for examination no tumor can be detected, and no other sign or symptom is present which definitely localizes the disease in the stomach. Consequently, when emaciation is present, carcinoma of the stomach may be easily confounded with Addison's disease, pernicious anemia, forms of tuberculosis, chronic malarial poisoning, certain dynamic affections of the stomach, displacements of the stomach, non-malignant obstruction of the cardia and pylorus, gastric ulcer, and chronic gastritis. (For the differential diagnosis between cancer of the stomach and ulcer, anorexia nervosa, nervous vomiting, myasthenia, displacements of the stomach, and non-malignant obstruction of the cardia and pylorus see the articles on these diseases.) There remains for consideration here the constitutional cachectic diseases and chronic asthenic gastritis.

Addison's disease is manifested by the bronzed skin, extreme weakness of the muscular system, disturbances of the stomach, and a form of emaciation in which the body fat may be partly conserved, as in cancer. Certain forms of tuberculosis run a slow progressive course without recognizable local deposits. Tuberculosis is also common in cancer of the stomach. Chronic malarial poisoning may develop without chills, and lead in a number of months to pronounced emaciation and cachexia. Other particular cases of chronic disease develop without very definite symptoms, and may resemble closely the atypical cases of cancer, which are not few in number. The differentiation can be made only by very careful search for distinctive signs and by counting the probabilities. In obscure cases of suspected tuberculosis local deposits of the disease should be sought in every part of the body. Sometimes the only detectable local lesion is tubercle of the

choroid or a laryngeal or nasal ulcer. The tubercle bacillus should be sought for in the urine when not found in the sputum. The absence of reaction after the use of Koch's tuberculin is decisive. In malarial cachexia the spleen is large and the crescent plasmodium is likely to be present in the blood, and the proper study of the blood would also detect a grave anemia. The other set of differential signs are those which are usually present and more or less characteristic of cancer. Here must be taken into consideration all the subjective and objective signs enumerated in the description of cancer of the stomach, particularly the functional, bacteriological, and anatomical signs. The weighing of all the evidence accumulated by careful study may, in particular cases, leave room for doubt, for any of these diseases may coexist with cancer of the stomach.

Cancer of the stomach without a palpable tumor may be confounded with chronic gastritis, particularly when the latter is accompanied by emaciation.

There are many features which differentiate cancer of the stomach from the asthenic form of chronic gastritis, except in the very early stage of cancer, when the two diseases may be so nearly alike as to leave little opportunity for a plausible guess. Cancer begins after thirty, often in persons with good and unconscious digestion, suddenly and without palpable cause. Gastritis begins after an acute attack due to some dietetic or drinking fault, or slowly and irregularly in a stomach which has often shown its weakness, or secondarily to a disease of some other organ. The subjective symptoms of gastritis, except the morning nausea and vomiting of mucus, are digestive. Those of cancer may be worse during digestion, but are often present day and night. The digestive symptoms of gastritis are much worse after solid than after liquid food, and a carefully selected meal may often be digested without discomfort. In cancer the heaviness and fullness recur regularly, and after all sorts of food. The appetite in cancer diminishes, is lost, never recovered, and often ends in disgust for meats and fats. In chronic asthenic gastritis the appetite is irregular, sometimes good, sometimes bad, and often there is a desire for spicy or sour articles. Vomiting is rare in chronic asthenic gastritis, and when present usually consists not of food, but of a little mucus, which may be stained with fresh blood. In cancer vomiting is much more frequent, alimentary, smelling of organic acids, and colored like coffee grounds with blood which has been long in the stomach. In chronic asthenic gastritis the hemorrhage is an accident

of sounding or of retching; in cancer it is often slow, not due to traumatism, and is sometimes profuse. Chronic asthenic gastritis is a painless disease, except sometimes in its atrophic stage. Cancer may be painful during the period when the stomach is functionally active, and also when it should be resting. The one develops slowly, with remissions and subjective intermissions, without a tumor, without cachexia, and without hemorrhages; the other progresses rapidly and continuously, with only short intervals of improvement obtainable by treatment. Chronic asthenic gastritis extends through years without producing cachexia; cancer is rapid and progressive in its evolution. Secretion diminishes in both, but free hydrochloric acid may reappear in chronic gastritis. The motor function is long perfectly maintained in chronic asthenic gastritis; in cancer stagnation occurs, as a rule, early, and gastric retention may not be long postponed, even when the neoplasm is situated on the body of the organ. Fermentation is an accident in chronic asthenic gastritis; it occurs very frequently, and often early, in cancer, is chiefly lactic, and accompained by a characteristic bacillary growth. In chronic asthenic gastritis the stomach is empty in the early morning before breakfast, or contains only a little fluid, which is rich in mucus and chlorids, and is about neutral in reaction. In cancer the stomach often contains residual fluid in the morning, which may be richer in combined HCl than the filtrate of the test-meal contents; or which may possess a high acidity due to lactic acid, or which may be colored dark brown by blood. The anatomical signs may reveal the presence of the neoplasm, but a piece of the mucous membrane in the washings showing only gastritis would not exclude cancer.

Chronic hypersthenic gastritis is readily distinguished by its irregular, stormy, and intermittent beginning, by the evolution of the digestive symptoms in relation to secretion and to the quality of the food, and by the invariable presence of free HCl after a test-breakfast. The two diseases, in their subjective and objective signs, bear little resemblance; unless, as very seldom happens, the neoplasm is engrafted in an old ulcer. The presence of a tumor is against gastritis, unless the gastritis be accompanied by an ulcer. If the clinical history, duration, and evolution do not differentiate them, the diagnosis is left in doubt, but with an exceedingly strong presumption against carcinoma. If ulcer can be excluded, carcinoma goes with it. Simple ulcer and chronic hypersthenic gastritis may

become complicated by engrafted cancer, which converts them into malignant and progressive diseases.

Prognosis.—Cancer of the stomach is invariably a fatal disease. Life may be made more comfortable and slightly prolonged by careful management. Early and radical operation may give the patient a few months, or, possibly, one, two, or even five years. The healing art can afford only a little more comfort than unaided nature would give the hopeless patient.

Treatment.—The treatment of carcinoma of the stomach is medical and surgical. The medical treatment is only palliative, but the surgical treatment may be employed not only with a view to give temporary relief and to prolong life, but also, in the early stage, with a barely possible hope of a permanent cure.

The **medical treatment** is hygienic, symptomatic, and protective. Every means should be employed to preserve the strength and weight and to diminish the excessive nitrogenous waste. Physical and mental rest are imperative, for the organism has little power for repairing its losses. Attention should be given to the skin and to the nervous system, with a view to maintaining their functions and vigor by hydrotherapy, pure air, sunshine, and pleasant surroundings. Digestive hygiene is also useful, and is briefly comprised in bodily rest before and after meals, in favoring the stomach, and in protection of the intestines.

It may well be doubted that any drug arrests or influences beneficially the growth of the neoplasm, as has been claimed for condurango, chlorate of soda (Brissaud), sulphate of anilin (Fay), and pyoktanin (Maibaum). Condurango is an excellent bitter which may often be employed with advantage, and methylene-blue in some cases seems to be of value. The methylene-blue (Merck) should be given in a capsule of three to five grains daily, and a little powdered nutmeg should be combined with it to correct its slightly irritant action on the urinary tract. Marcus Fay claims that anilin sulphate delays metastasis and cachexia, and relieves the pain better than opium. Given internally, in one to five grains a day, its absorption lasts about two hours, when the nails and lips become blue, and after several days' administration the urine becomes reddish. In full doses it produces vertigo, shivering, dyspnea, fainting, and tonic contraction of the involuntary muscles. None of these drugs, it must be confessed, is of much value, except condurango as a bitter and methylene-blue, or phenacetin, or codein, or opium and belladonna to relieve the pain.

But the medical treatment does not consist in the vain search for some specific, nor in efforts to arouse the depressed functions of the stomach. The treatment of the stomach should be protective and not excitant, irritation of all kinds only doing harm. But the maintenance of the appetite and of the motor function is well worthy of attention, and may be best accomplished by a combination of the infusion of condurango bark and hydrochloric acid with strychnin. The prescription should be ordered half an hour before meals, and is most valuable when lactic acid fermentation is present. Hydrochloric acid (but it sometimes is not well borne) may be ordered in repeated doses during the period of gastric digestion.

Pain is often distressing, and demands relief. When the pain is severe, there should be no hesitation in giving codein or morphin, hypodermically or by the mouth, after milder analgesics have failed. There is nothing to fear from a possible opium habit when the patient is already the victim of a malignant disease. Vomiting may require the usual efforts to control it. No one procedure does more good than lavage. A wet compress, covered with a rubber coil, through which hot water flows, the application being made half an hour before the meal and kept on during the period of digestion, may be beneficial.

To control fermentation and to remove the products of retention, lavage acts better than any other remedy, but it should be employed, when the disease is advanced or when there is hemorrhage, with the greatest care or not at all. This is particularly true when the neoplasm involves the body of the stomach. But in cancer of the pylorus and in infiltrating scirrhus it may be used without danger, but not more than a pint of water should be allowed to flow in before beginning to withdraw it; the danger of perforating the degenerate wall by overdistention is thus avoided. The lavage should be performed thoroughly in the morning, an hour before breakfast, and the hydrochloric acid tonic should be administered a half hour later.

The diet should be regulated so as to favor the stomach, to protect the intestines, and to maintain nutrition as long as possible. An exclusive or reducing diet in this disease is radically wrong, and the food selected should not excite or irritate the stomach, remain long in it, easily ferment, or be indigestible by the intestines. Milk seldom agrees well, except in the early stage of some cases where stagnation and fermentation are slight. In the stage of gastric retention it

only adds fuel to the flame. Consequently, finely-divided tender meats, lean fresh fish, and white of egg must usually be depended upon to furnish the nitrogenous needs of the body, but should not be given in excess with the vain hope of covering the excessive nitrogenous waste. Fat, in the form of fresh butter or a good emulsion of cod-liver oil, is valuable and digestible in moderate quantity. Meat juice and clear vegetable soups may be prescribed in order to furnish the requisite quantity of salts. Meat jellies often agree well and supplant the sweets, which must be excluded. Very thoroughly cooked whole wheat, with all the bran removed, and purées of vegetables digestible by the intestines usually agree well when the stomach is kept clean by lavage and the hydrochloric acid tonic is given. Supplementary rectal feeding should be employed early, and not held back as a last resort, when it is too late to be of much value. Alone, it exerts little influence on the progressive inanition of carcinoma.

The **surgical treatment** is palliative and prolongs life. In cancer of the cardia gastrostomy should be performed as soon as the patient is no longer able to swallow enough food to nourish the body. An attempt to dilate the obstructed cardia by means of esophageal sounds or dilators is more likely to be injurious than beneficial, on account of the swelling and inflammation excited and the danger of perforation. Little can be said in favor of the esophageal cannula. Where retention occurs above the obstruction, the constant irritation causes the neoplasm to grow more rapidly, and gastrostomy might be performed early in order to avoid this effect. But, as a rule, gastrostomy should not be performed while the body can be nourished by combined oral and rectal feeding.

The most frequent operation for cancer of the body of the stomach and of the pylorus is gastro-enterostomy. This is only a palliative operation, which improves the motor function without perceptibly increasing secretion. The operation is often followed by a remarkable improvement in nutrition, and by the subsidence of the inflammatory swelling around the neoplasm. It is the best palliative operation, and should be performed when retention renders it no longer possible to sufficiently nourish the suffering patient.

Pylorectomy is an effort to produce a radical cure, but thus far it has failed. It is the preferable operation when the neoplasm is pyloric, without adhesions, enlarged glands, or metastasis, and when the operation can be done in sound tissue wide of the zone of extension. In suitable cases a

respite is obtained until the tumor recurs, and gastro-enterostomy may be done after the recurrence, to prolong life.

The mortality of pylorectomy for cancer varies from 55 to 27 per cent. When adhesions and complications exist, the mortality is much higher (60 per cent.) than in properly operable cases (25 per cent.). The reports of Billroth's clinic from 1880 to 1894 (Hacker) give 19 deaths in 42 operations; but only 4 of the last 16 cases died. The mortality of pylorectomy done by experienced surgeons does not differ materially from that of gastro-enterostomy. The latter operation appears to give greater immediate relief; and, with very few exceptions, the patients have lived as long after it as after pylorectomy. Kocher, Czerny, and Ratimmow report cases in good health from four to eight years after pylorectomy. No radical cure has been obtained in Billroth's clinic, although one patient lived more than five years.

CHAPTER IV.

THE DISPLACEMENTS OF THE STOMACH.

Transposition of the stomach is a very rare condition, and one which can easily be discovered on physical examination. The fundus is transposed to the right, and the pylorus lies in the left hypochondrium. The other parts of the digestive tube and its accessory glands are correspondingly changed in position. This is an anomaly of development, not a disease, and requires no further mention.

The pathological displacements of the stomach, particularly frequent in women, are very numerous in their anatomical details. But all of them are deviations from the three grand clinical types—upward displacement, lateral displacement, and total descent.

The abdominal cavity, formed in part by pliable walls, is subject to the action of atmospheric and other external pressure, and the organs and viscera contained within it readily change their form. The stomach is attached by ligaments to the liver, spleen, diaphragm, and transverse colon, and is continuous with the esophagus and duodenum. Consequently, the displacements of the stomach are accompanied by changes in the form of the abdomen and in the form and relative position of neighboring organs. Its attachments, also, are ren-

dered lax or are stretched, thus causing displacements of attached organs and disordering the blood- and lymph-circulation. These results often interfere with the nutrition of its coats and with its functions. The interference with nutrition by compression and by traction may be localized and circumscribed, and a strong predisposition to ulcer may thus be created. The new relations produce new points of contact and new directions of least resistance, and consequently the viscus is liable to undergo particular changes in form. The churning and evacuation of its contents must be done in unusual and unfavorable circumstances. The evacuation of the stomach may be specially difficult on account of the traction brought to bear on the beginning of the duodenum and the pyloric region, and on account of the constriction of the duodenum at its first point of firm attachment. The necessity for increased work at a mechanical disadvantage entails either compensatory hypertrophy of the muscular layer or motor insufficiency. The clinical forms of displacement are three in number—upward, lateral, and downward.

1. **Upward Displacement.**—The upward displacement of the stomach can occur only in the left concavity of the diaphragm. The part of the stomach to the right of the cardia can not be displaced upward, for the organs above it are solid and fixed. The upward displacement of the stomach is less frequent than the other forms of displacement, but it occurs much more frequently than is generally recognized.

Etiology.—The fundus of the stomach may be situated abnormally high when the left lung is collapsed (atelectasis, sequel of left pleurisy), or the stomach may be forced upward by a large abdominal tumor. But the most frequent cause of this form of displacement is compression and arrested development of the trunk on a line which runs across the abdomen near the umbilicus and below the liver, the splenic flexure of the colon, and the spleen. The costal arch is slightly narrowed, and the lower four or five ribs are forced far inward, so as to make the smallest part of the waist nearly on a line with the iliac crests.

Clinical Description.—Upward displacement of the stomach may be a latent disease, or, at least, only insignificant subjective symptoms may result from it; but in some cases the distress occurs in paroxysms, and in others the disturbances are persistent. The symptoms vary greatly, and bear a relation to the manner in which the displacement has been produced. There may be only a slight sense of fullness in the

left hypochondrium after meals, or great difficulty may be experienced in vomiting, in belching, or in eructation, the cardia being drawn upward and to the left and the esophagus being obstructed at its point of passage through the diaphragm so as to prevent the exit of the contents of the stomach. The upward displacement of the fundus and its distention with gas may produce shortness of breath, palpitations, arrhythmia, left intercostal neuralgia, and precordial pain. These symptoms may occur in paroxysms after eating and after exertion during the digestive period, but are most frequent in the evening after the chief meal of the day. When the displacement results from the creation of the long and low waist, the same gastric symptoms may be present, but to these are added symptoms due to the displacement and to compression of the colon; the transverse colon is shortened and falls into a V-shape, the splenic flexure is made more acute, the hepatic flexure is prolapsed or forced inward, and the colon is compressed against the spinal column and by the tips and borders of the ribs. There results from this compression and deformity a series of troubles—constipation, stagnation, ulceration, pseudomembranous formation, points of peritonitis, together with all their local and general effects.

Objective Signs.—In upward displacement of the stomach there may be but little alteration of the position of the greater curvature; but, as a rule, only the pyloric end of the stomach lies in the epigastrium, the pylorus being displaced transversely to the left, and the greater curvature lying so high as to create the impression that the stomach is abnormally small. Gastric tapping-splashing can not be elicited for the reason that so little of the stomach lies in the epigastrium. On percussion, the superior border of the fundus is abnormally high, and, usually, abnormally broad; and these abnormalities exist when the stomach is only moderately distended with air or gas. It is often possible to produce succussion splashing, and the location of the sound will roughly reveal the situation of the stomach, which may be determined with precision by percussion after inflation and after filling the stomach with water, and by electric illumination. The heart is sometimes displaced to the right by the distended fundus.

Treatment.—The treatment comprises the removal of the cause so far as possible; the prevention of the accumulation of a large quantity of gas in the stomach; the avoidance of heavy meals; and rest, with the clothing loosened, during the period of digestion. The alkaline carbonates and effervescing

drinks do harm, and the diet and medication should be so ordered as to obtain rapid evacuation of the stomach and to prevent spasm of the pylorus. The severe respiratory and cardiac paroxysms are relieved, as if by magic, by the withdrawal of the gas from the stomach by means of the stomach-tube. Antispasmodics and carminatives are far more valuable than stimulants and anodynes. Digitalis, strophanthus, and similar heart stimulants increase the palpitation and the arrhythmia. The increase of abdominal tension should be avoided, and intestinal flatulency should be controlled.

2. **Vertical or Lateral Displacement.**—In vertical displacement of the stomach the position of the cardia and of the line marking the superior border remains as in health. The part of the stomach in the left concavity of the diaphragm, and above a plane cutting across the body on a level with the cardiac orifice, is unchanged in its form and position. The changes characteristic of vertical displacement occur below this line. The greater curvature is displaced downward to the left, cutting the costal border near the tip of the tenth cartilage. The lesser curvature becomes straighter or is bent near its middle into almost a right angle, accordingly as the pylorus is more or less displaced. The anterior and posterior surfaces of the stomach face more directly forward and backward. The axis of the pyloric end runs transversely, or downward toward the right iliac fossa, or more directly upward, the variation corresponding with the particular form which the stomach takes. The greater part of the grand axis of the stomach is almost vertical. The relations of the stomach are changed and its form is greatly modified, particularly the transverse diameter. Such are the general characters of vertical displacement. The special anatomical forms will be more minutely described in the paragraphs on the pathological anatomy, and should be carefully studied, with a view to facilitating the solution of many of the puzzles of differential diagnosis.

Etiology.—Vertical displacement of the stomach is rare in men but quite frequent in women, who seem compelled by fashion to deform their waists in slavery to a false conception of the beautiful. The disease may be a legacy of infancy or of fetal life, the stomach remaining in the position in which it was held by the relatively and excessively large liver of this period. The stomach may also be vertically displaced by enlargement and tumors of the liver, and by other tumors and by pus collections to the right of the lesser curvature; but by far the most common cause is the corset. In the verti-

cal displacement produced by the corset the line of greatest compression runs across the liver, the first portion of the duodenum, the pylorus, and the spleen. The costal arch is very narrow, but the base of the arch may be broad and the costal borders curved outward and forward. The surface of the liver is grooved by the ribs, and the gland is compressed from side to side and elongated downward. The pylorus is obstructed by the compression, as are likewise the duodenum and the cystic and common ducts. The pyloric end of the stomach dilates downward below the constriction, and the stomach may be made bilocular, the area of constriction passing between the spleen, the depressed ribs, the left lobe of the liver, and the vertebral column.

Genesis and Pathological Anatomy.—The principal parts in the development of vertical displacement of the stomach are played by the left lobe of the liver and by the constriction of the waist in a particular manner. The compression of a tight corset worn during the period of puberty arrests the development of the constricted part of the body. All the diameters of the abdominal cavity throughout the compressed region are shortened and their increase during the period of development is prevented. The compression of the corset, however, differs from that of a cord or a narrow band, and extends from near the cartilages of the eighth ribs to within a short distance of the breasts. The costal arch is narrowed, except near the base, where the costal border curves rapidly outward and forward. The results of the cylindrical compression are displacement and deformity of the organs brought within its influence. As there can be no lateral expansion, the respiratory movements of the organs of the abdomen are directly up and down. The solid organs, such as the liver, push what is movable before them, and the deforming pressure of the liver and the distention of the gastric wall occur downward, in the direction of least resistance. The fundus of the stomach is above the area of compression; but lower down, the costal wall, the spleen, the liver, and the stomach bear the brunt of the pressure. It is here nearly on a level with the ensiform process that the waist is smallest, and the liver is often marked by a furrow running across its anterior surface. The stomach yields its place to the solid liver, there being only sufficient room (provided the corset has been put on early in life and worn tight) between the anterior abdominal wall and the vertebral column for the left lobe of the liver and the pancreas. The lesser curvature of the stomach, which is compressed between the liver, the

spleen, and the ribs, is forced to the left and downward, and runs along the border of the left lobe. If the spleen and liver be enlarged, the compressed part of the stomach may be reduced to the size of the small intestine. Nearly the whole of the deformed stomach thus lies vertically displaced to the left of the left parasternal line, and it may be bilocular.

There are three typical anatomical forms of vertical displacement of the stomach—the angular, the fish-hook, and the straight.

The *angular form* is quite common, and is the result chiefly of the form and size of the left lobe of the liver. The lesser curvature, and with it the body of the stomach, are displaced outward to the left and into the left hypochondrium. The pylorus is about on a level with the junction of the middle and lower thirds of the line joining the umbilicus and the xiphoid process, either on the median line or about an inch to the left of it. The pyloric end, forming one side of the angle, runs transversely across the abdomen and close along the lower border of the left lobe of the liver, by which it is not covered. The pancreas may form the limiting and fixing upper wall, the left lobe being a little higher up, with its lower border curved backward on a transverse axis. At the point where the stomach passes beneath the left costal border a constriction often exists, which is yielding and not cicatricial. The greater curvature passes the costal border low down and far to the left. Along and partly covered by the costal border often lies the distended and most capacious part of the deformed stomach. The other side of the angle, represented by the remainder of the long axis of the stomach, passes vertically through the left hypochondrium. The angle, instead of being sharply formed, is sometimes cut off by a short curve.

The *fish-hook variety* is a very serious form of displacement. The pylorus usually remains in its normal position, but the orifice faces almost directly upward. The pyloric extremity of the stomach rises almost vertically over the head of the pancreas, and is continuous with the duodenum, which runs upward and to the right, joins its fixed part at an angle, and produces a constricting kink. The lesser curvature runs below both the left lobe of the liver and all the pancreas but the head. The greater curvature sweeps by the navel and passes under the costal border near the tip of the tenth rib. This curved part of the hook is often dilated, and the greater curvature may extend downward and to the right across the median line. The sharp convexity of the dilated pyloric part points to the right iliac fossa. On account of this particular

form of the stomach its muscle works at a great disadvantage in its efforts to overcome the obstruction in the duodenum. The vertical part of the stomach, pushed far into the left hypochondrium, is often compressed between the overlapping left lobe of the liver and the spleen, and the transverse diameter of the stomach at the point of compression may be reduced to 1½ or two inches.

In the *straight form* the duodenohepatic ligament is elongated, and the pylorus lies very close to the umbilicus. The lesser curvature bends slightly on its way to the cardia. Nearly the whole of the long axis of the stomach is vertical, and the body of the organ, on account of the particular deformity of the liver which causes the displacement, is usually but slightly compressed. This is the least frequent of the three varieties.

The vertically displaced stomach may descend in the abdomen, and the upper limit of the fundus may be lowered. Associated with vertical displacement of the stomach are deformities and displacements of other abdominal organs. The liver rotates on a transverse axis, or descends in the abdomen, or is pressed out of its normal shape. The right kidney is more or less movable. The spleen is displaced and deformed. There may be splanchnoptosis or enteroptosis, particularly if the abdominal wall is flabby.

Clinical Description.—Vertical displacement may exist without producing a local or general subjective symptom. It is a disease which is often without an expression. From puberty to old age digestion may be good and the general health excellent. This latency constitutes its seemingly harmless nature and also its serious danger. A vertically displaced stomach is a constant menace to health.

There can be no doubt that a vertically displaced stomach does its work at a great disadvantage, and the motor function upon the integrity of which health depends may easily become insufficient. The organ is in a state of unstable equilibrium.

One class of subjective symptoms is due, not to the displacement of the stomach alone, but to the combined effect of the changed relations and deformities of the abdominal organs. There is sometimes localized peritonitis, traumatic in its genesis. This is manifested by local pain and tenderness, and by constitutional depression, most often mistaken for visceral neuralgia. The sympathetic nerves are irritated by being stretched and compressed, often producing local discomfort, which is most marked after walking and jolting exercise; and dragging sensations may be experienced about the

loins. The abdominal sympathetic nerve may be so irritated as to cause neurasthenic symptoms. Neurasthenia is a common expression of a developing or unsuspected displacement of the abdominal organs, including vertical displacement of the stomach.

But the displacement and the constriction that caused it do more: the blood- and lymph-circulations are disordered. The blood-vessels running in the folds of the ligaments are stretched, compressed, and distorted, here producing stasis, there causing anemia. Furthermore, the intra-abdominal tension is modified, and the beneficial effect of respiration on the circulation of the abdomen, particularly that of the lymph, is diminished. The result is that these patients often emaciate, age early, become anemic, lose energy, and are morose, melancholic, and sometimes irritable. This change of character and disposition is often a revealing sign. The characteristics of the anemias produced by the displacements of the stomach are that they are rebellious to treatment, are not much benefited by iron or arsenic, but are cured by rest in bed and proper alimentation; and this is likewise true of the neuralgic, neurasthenic, and psychic symptoms produced by the disturbed equilibrium of the abdomen.

The fish-hook form of vertical displacement may, as has already been stated, produce duodenal obstruction. The symptoms are, then, those of the stages of compensation, stagnation, or retention, as described under pyloric obstruction; but the distinctive sign is the discovery of a stomach vertically displaced and assuming this particular form. A constant danger of the vertically displaced stomach is motor insufficiency.

Objective Signs.—The physical signs are characteristic, for they reveal the particular form and position of the stomach which constitute the disease. An important and suggestive sign furnished by inspection is the corset-waist. The costal borders are compressed, and just below the ensiform process is the junction of the two cones, with their bases facing up and down, the patient being viewed from in front. The lower costal borders are somewhat everted. The arch formed by the costal borders is narrowed, the upper portion of both borders approaching nearer to the median line. The epigastrium is flat, and the lower part of the abdomen is prominent. The lateral expansion and development of the cavity inclosed by the false ribs have been arrested. Naturally, this suggestive form of the abdomen is lacking when the displacement of the stomach has been caused by enlargement

of the left lobe of the liver or by tumors of the right hypochondrium.

More conclusive are the signs which reveal the form and position of the stomach. The abdomen should first be examined when the stomach is empty. The organ is next inflated. It will first be noted that the epigastrium has not been distended by this procedure, but retains its flat form. If the abdominal wall is lax, the small portion of the stomach uncovered by the left ribs may be seen outlined as a prominent ridge. On percussion, the upper limit of the fundus of the stomach will be found in its normal position, and the greater tuberosity will reach as high as the fifth interspace. The line of the greater curvature runs far to the left, emerges from under the ribs near the cartilage of the tenth rib or a little higher, and ends at the pylorus, which may be on the median line, to the left or the right of it, or in its normal position, as described under the pathological anatomy. The lesser curvature is displaced downward and to the left, and is located by inspection, palpation, and percussion along the border of the left lobe of the liver. In the median line of the epigastrium only the left lobe of the liver and the pancreas are interposed between the anterior abdominal wall and the spinal column. The aorta can here be easily felt, and its pulsation is often visible. A glass of water should next be administered, and by the aid of the splashing sounds confirmatory evidence of the position and form of the stomach can be obtained. The water and gas can often be felt and heard bubbling through the pylorus. The position of the curvature and of the pylorus reveals the angular, fish-hook, and straight varieties of vertical displacement.

It will be noted that the transverse diameter of the stomach near the level of the lower border of the left lobe of the liver is very small. The stomach also extends low down in the abdomen, and a great part of it lies in the left hypochondrium. On account of its position and form and the lines of compression, respiratory gurgling is often produced when the organ contains fluid and air. This symptom is a shock to modesty, and may be arrested by changing the position of the body, by making the respirations shallow, by loosening or tightening the clothing, or by compressing and lifting the left side of the abdomen. The noise is produced by the forced passage of fluid and gas from one pouch of the stomach into another one separated from it by a constriction. The constriction is often produced by the border of the ribs and by the colon, while pressure is made alternately on the two

pouches by the up-and-down movements of the stomach during respiration. This sign is most common in vertical displacement of the stomach, but is not pathognomonic.

There are no abnormal functional signs of vertical displacement of the stomach. In only one variety—the fish-hook deformity—is the evacuation of the stomach interfered with, and motor compensation may be perfectly established. In many cases of vertical displacement it is impossible for the patient voluntarily to belch or to vomit. The desire may be imperative, but the effort to do either is often a failure.

The abnormal functional and bacteriological signs found in vertical displacement of the stomach are due to complications. The most frequent of these complications are myasthenia and gastritis.

Prognosis.—The vertically displaced stomach, though working at a disadvantage, often performs its work well. With proper digestive hygiene the patient may go through life without digestive trouble. But it constitutes a weakness, a predisposition to disease, and a danger. When it exists, the stomach is more liable to disease, and the cure of such engrafted disease is rendered difficult.

Treatment.—Much can be done in the prevention of displacement of the stomach by protection of the waist against undue compression. Custom intervenes at an early age and enforces the arrest of the natural development of this part of the female body. No great harm need be done if the lacing be held within limits. A corset which is of the proper size can be fastened during gastric digestion at the end of an ordinary expiration without drawing on either side, and is harmless when this rule is followed from youth. This matter should receive the personal attention of mothers who value the health and beauty of their daughters more highly than an unnaturally small waist. The skirts should be supported from the shoulders, and the corset and clothing should never be pushed down after fastening, with a view to lengthening the waist.

After the displacement is produced, the excessive constriction of the waist should be removed, and combined arm and breathing exercises prescribed in order to widen the costal arch and to develop more space in the epigastrium. The abdominal muscles should be strengthened by exercises, massage, and faradism, and the lower abdomen should be snugly supported by a bandage. The abdominal support should always be tightened from below upward before the

corset is put on, and, in severe cases, while the patient is in the knee-chest posture.

The treatment of a complicating disease is the same as when the stomach is in its normal position. But if myasthenia and the secondary anemia require a remedy in addition to those ordinarily employed, rest in bed should be prescribed. In the mild form of myasthenia and in mild anemia the patient should recline, with the clothing loosened, during the greater part of the period of gastric digestion. In the severe form of myasthenia with stagnation, in myasthenia with retention, and in advanced and grave anemia, a methodical cure with uninterrupted rest in bed should be employed.

3. **Gastroptosis, or Total Descent of the Stomach.**—Gastroptosis is the most frequent displacement of the stomach, and is usually associated with the displacement of other abdominal organs. The whole stomach is prolapsed, and lies nearly transversely in the abdominal cavity. The pylorus may remain near its normal position, but it is commonly lower down. The horizontal part of the duodenum is the first fixed point, and it is here that angular obstruction is likely to occur. The cardia also descends and lies on a level with the body of the twelfth dorsal vertebra. The fundus of the inflated stomach is below the fifth rib, and the lesser curvature runs across the abdomen in the lower half of the line joining the umbilicus and the ensiform process. The point where the greater curvature crosses the costal border is further down and further to the left than it is normally located, and the lower limit of the stomach runs across the abdomen and the median line below the umbilicus, sometimes as low as the pubic symphysis. The pyloric antrum or lesser tuberosity is commonly to the right of the median line, and sometimes is larger than it should be. The stomach is not simply enlarged, for there may be no increase in capacity; but the whole organ descends in the abdomen, and this total displacement downward constitutes the gastroptosis.

Etiology.—The abdominal viscera are held in position by ligaments and attachments and by the elasticity of the abdominal wall. The stomach is supported, also, by the colon and other organs in the abdominal cavity beneath it, and may be displaced by compression, by traction, and by its own unsupported weight when full.

The greater tuberosity of the stomach lies in close contact with the diaphragm, and may be displaced downward by certain diseases of the thorax, such as left pleuritic effusions, emphysema, or pericardial effusion. The downward displace-

ment of the diaphragm, particularly if the abdominal wall is lax or if abdominal tension is reduced by emaciation and by emptiness of the intestines, disturbs the equilibrium of the abdomen, and may inaugurate a series of displacements which will eventually lead to the permanent descent of the stomach. But often the displacements are temporary, and the abdominal equilibrium is restored after the diaphragm returns to its normal position.

Every severe acute disease leaves the digestive tube and the entire system enfeebled. Typhoid fever, severe influenza, pneumonia, and acute tuberculosis are particularly active in this respect. Overloading the motor-insufficient stomach during convalescence drags the organ down against the feeble resistance offered by its weak ligaments. Moreover, the acute infectious diseases enlarge the liver and spleen and weaken all the ligaments. The equilibrium of the abdomen is thus disturbed, and the displacement of one organ after another may eventually produce gastroptosis.

There is no doubt that weak muscular and fibrovascular systems may be inherited or are acquired by disease during infancy. Many persons go through life afflicted with weak hearts, weak digestion, weak lungs, torpid liver, and bad portal and lymph circulations. This inherited or early acquired weakness may affect particularly the peritoneum, and may constitute a particular fibroid dyscrasia; it is commonly associated with gastro-intestinal myasthenia and a consequently variable intra-abdominal tension. Be the pathogenesis what it may, there is no question that gastroptosis is frequent in those cursed by such an inheritance or by this legacy of abdominal disease in early life.

Gastric retention is also a cause of gastroptosis. The weight of the full stomach pulls the organ down, and its descent meets with less resistance from the empty intestines and from the greater space in the abdominal cavity resulting from the accompanying emaciation and utilization of the deposited tissue fat. It is common enough to find gastroptosis associated with obstructive and myasthenic retention, and in some of these cases the total descent of the stomach is a result.

A very flabby abdominal wall associated with low intra-abdominal pressure is another cause of gastroptosis. Pregnancy, a short lying-in period, sudden and extreme emaciation, the removal or absorption of ascitic fluid, and the removal of large abdominal tumors are the chief causes of the flabby, collapsing abdomen. The organs and viscera, unsupported

in the erect position, drag on their ligaments and attachments, and descend eventually into the lower abdomen.

Tight lacing may produce upward, lateral, or downward displacement of the stomach, according to the location of the line of greatest constriction and to the shape of waist produced by the corset. The vertically displaced corset-stomach may undergo prolapse. But when tight lacing, aided often by other causes of gastroptosis, produces total descent of the stomach, the line of greatest constriction runs across the convex surface of the liver, between the sixth and eighth ribs, and across the abdomen near the ensiform process. The liver, the right kidney, the pylorus, the colon, and the spleen are simultaneously displaced downward, and the whole stomach is pulled and pressed out of the position which it normally occupies. The waists of gastroptosis, of lateral displacement, and of upward displacement may be distinguished, in their typical forms, at a casual glance.

Displacements of the various abdominal organs usually occur in association, and displacement of one organ may be the cause of displacement of another. But it is certainly an error to make gastroptosis, as is sometimes done, a mere episode in the evolution of enteroptosis. Glénard maintains that the consequences of prolapse of the hepatic flexure of the colon are descent of the transverse colon, of the pylorus or the stomach, of the liver, and of the right kidney. That such is always the order of development can not be successfully defended. The vicious circle may begin with any of the organs, and the displacements may develop partly in consequence of common causes, partly as the result of changing relations of the organs and viscera, producing new directions of least resistance. The tendency is all downward, on account of the constriction above and of the insufficient support below when in the erect position. In two-thirds of the cases of gastroptosis the hepatic flexure of the colon is not prolapsed.

Clinical Description.—Gastroptosis is exceedingly variable in its expression. This variability is in keeping with the multiplicity of its causation, the constitutions of its victims, its numerous associated displacements, and the divers diseases of the displaced organs. But the course of the disease presents one pronounced characteristic—there is no tendency to get well, nor is permanent relief obtained without proper mechanical treatment.

In the beginning, for a longer or shorter period, digestive compensation may be maintained, and the symptoms be but

little influenced by the quality or quantity of the food. Later, during the period of gastric digestion, the patients often complain of a sensation of heaviness or fullness, which disappears in the recumbent position but reappears on standing. There is uneasiness, discomfort, and dragging sensations in the epigastrium and back; the patient grows weak, irritable, and pessimistic, and becomes an invalid without knowing any reason for it. The cause is discoverable only by physical examination.

The motor function of the stomach eventually becomes insufficient. There is gastric stagnation, often heartburn, flatulency, and severe pain. Stagnation in the colon is common, and the constipation may alternate with diarrhea or the stools may consist of hard lumps coated with mucus and mixed with fluid and gas. Certain foods increase the symptoms, which are now constitutional and local. Vinegar, acid drinks, wine, and milk particularly increase the digestive symptoms, the nervousness, depression, headache, and the insomnia of the second half of the night. Neurasthenic pains and tender points appear. The back aches and the lower extremities are weak. The patient restricts the diet, and loses weight and strength. These symptoms may continue, with exacerbations and remissions, for a number of years. Excessive uric acid formation is sometimes a symptom of the established disease, but more frequently the uric acid is deposited in the urine after standing without an excessive formation, the precipitation being due simply to diminished solvent power of the urine or to excessive conversion of the neutral phosphate of soda into the acid phosphate.

The stagnation eventually may be replaced by retention. The emaciation then increases, and the insomnia becomes complete. The intestines are more and more disordered, and enteritis membranacea, with alternating stenosis and stagnation, is quite common. The symptoms become more continuous in character and more independent of the digestive period and of the quantity and, to some extent, of the quality of the food. They are but slightly relieved by the recumbent position and by abdominal support. The dragging sensations, most pronounced during the developmental period, usually disappear in the course of the disease. Vomiting is very infrequent and exceedingly difficult, the flatulency can not be gotten rid of by belching, and constipation is usually obstinate. The propulsion of the contents of the digestive tube is made more difficult by the low abdominal tension, by the flabby abdominal walls, and by the stenosed fixed points of the digestive

tube, when they exist. The patient is nervous, weak, irritable, anemic, uricemic, and often neurasthenic, and suffers from catarrh of more or less all the mucous membranes. Fortunately, the disease seldom arrives at this advanced stage, and is often milder in its expression and amenable to proper treatment.

Symptomatology.—The digestive symptoms are variable, but present certain characteristics which should excite suspicion and lead to a careful physical examination of the stomach. The heaviness in the stomach after eating or drinking, the heartburn, flatulency, belching, and regurgitation, are symptoms of myasthenia. But rest in bed or rest during the period of gastric digestion causes them to disappear rapidly. The peculiar digestive discomfort and heaviness are relieved, even while the patient is in the erect position, by supporting the stomach, and they return as soon as the organ is released.

There are local pains independent of digestion and of the quality of the food. These pains are produced by stretching of the nerve filaments in the supporting ligaments, and, possibly, by a local peritonitis produced by a tear or detachment of the peritoneum. The latter is not a plausible explanation except when the displacement has been rapidly produced: as, for example, by traumatism or strain. The pain is sometimes severe, and is associated often with a peculiar local uneasiness or discomfort. The severe pain has two common locations—sometimes beneath the ensiform process, and sometimes near the left costal border just above the level of the umbilicus. The pain is excited by effort, by dancing, by jolting, often simply by a short walk. The severe pain may be accompanied by spasm of the diaphragm, by rapid pulse, by dilatation of the pupils, and by sudden changes in the color of the face. Some of these symptoms are doubtless due to the intense irritation of the abdominal sympathetic nerve.

Extreme and unaccountable weakness is a common symptom of gastroptosis, particularly when the disease is associated with a flabby abdomen and when it develops as a sequel of the puerperium. The mother's attention is fixed on the weakness and anemia, both of which increase so that the digestive disturbance is overlooked; while the physician is likely to suspect or incriminate the uterus, which, indeed, is often retroverted or prolapsed. But the chief trouble is the gastroptosis (sometimes splanchnoptosis) and the diminished

abdominal tension. No relief is obtained until these are properly treated.

Neurasthenia is one of the occasional results of gastroptosis. It presents no distinctive characteristics, except its association with the physical signs of the displacement, and its cure by the proper treatment of the gastroptosis.

Periodical headache is sometimes a symptom of gastroptosis, and seems to be due to the irritation of retained food and of excessive secretion. During the premonitory period the patient is nervous and restless, is constipated, and has often a little brow-ache in the morning. During the paroxysm the headache is severe, and is accompanied by precordial pain and great irritability of the sympathetic. The appetite is now completely lost. The attack ends with vomiting of food, some of which has been retained for two or three days; the vomit may be excessively rich in hydrochloric acid. This headache of gastroptosis is often confounded with migraine and adenohypersthenia gastrica. Its distinctive characteristic is the existence of the causative gastroptosis.

The objective signs are distinctive. On inspection, the form of the abdomen is often suggestive, the lower part being full or flabby and the epigastrium depressed. The contour of the stomach during the period of digestion can often be outlined on the abdominal wall by the practised eye. The lesser curvature runs across the abdomen lower than normal; the greater curvature is often below the navel, and may descend even as low as the symphysis. The greater curvature is not only lowered, but it extends much further to the left and further to the right of the median line than it should go.

If the stomach is empty, and, on percussion, the larger part of the semilunar space of Traube is dull, and if this dulness does not disappear when the stomach is inflated, the superior limit of the stomach is lowered. Care should naturally be taken to exclude pleurisy, pericarditis with effusion, consolidation of the lower lobe of the left lung, and other thoracic conditions which would diminish the resonance of this same area.

On inflation the curvatures can usually be located with accuracy. The descent of the lesser curvature and of the fundus are characteristic signs. The greater curvature can naturally be found below the umbilicus when the stomach is simply enlarged by gluttony, by pyloric or duodenal obstruction, in cases of advanced myasthenia, and when the stomach

is vertically displaced. The pylorus is usually lower than its normal position, and can be located by inspection after inflation and by palpation; it can be identified, also, by the periodical bubbling of the contents of the stomach through it. The epigastrium above the lesser curvature is dull on percussion, and the pancreas and left lobe of the liver can often be felt lying over the vertebral column and over the pulsating aorta. There often exists a painful, sharply limited, tender and epigastric point like that of ulcer; but a distinguishing sign is the absence of the stomach beneath the tender point of gastroptosis. In extreme gastroptosis with low abdominal tension, the epigastrium may possibly be occupied by coils of the intestines. Electric illumination of the stomach often produces a characteristic picture, but is not required for making the diagnosis. The other physical signs locate the stomach with greater ease and certainty.

The prolapsed stomach is not so closely associated with the movements of the diaphragm as when in its normal position. It often moves but little on either inspiration or expiration, is easily fixed during expiration, and can be grasped and readily moved about in the often flabby abdomen.

The left lobe of the liver and the sympathetic ganglia will often be found sensitive to pressure. This is in part due to the ease with which firm pressure can be brought to bear almost directly. When associated with the severe pain of effort beneath the ensiform process, and with neurasthenic tender points, the inexperienced may be led to suspect an ulcer.

The functional signs are in no manner characteristic of gastroptosis, but are of value in the revelation of complications and in the regulation of the diet and treatment. In the same respect the bacteriological and anatomical signs may be useful.

Differential Diagnosis.—The prolapsed stomach may become diseased in the same manner as when in its normal position. Associated with it may be found any of the anatomical diseases of the stomach and many of the dynamic affections. The diagnosis of the gastroptosis rests exclusively on its physical signs, and its causal relation to the associated disease often can not be determined. The clinical history may contain some distinctive features in favor of the priority of the displacement of the stomach, which, as may be expected, is very favorable to the development of other gastric troubles. The solution of the problem, however, is of little practical

moment, for the treatment must be that of the two diseases combined.

But gastroptosis is liable to be confounded with myasthenia, and with pyloric and duodenal obstruction. There are several signs which, although not conclusive, are in favor of gastroptosis. The flabby and prominent lower abdomen, the constricted waist, and the development of the gastric trouble as a sequel of pregnancy should create suspicion of the displacement. The great relief afforded, particularly during digestion, by the proper support and gentle compression of the abdomen below the umbilicus is another valuable differential sign. The existence of a movable right kidney, of a displaced and deformed liver, of a displaced and chronically inflamed colon, constipation, scybala coated with mucus, membrane-like shreds, alternating enterostenosis and dilatation, are most common in association with gastroptosis. But none of these signs is conclusive, and the diagnosis can only be made sure by the accurate delimitation of the stomach, demonstrating the total descent of the organ, which is made clear by the position of its superior border.

In long-standing cases of myasthenia, particularly in the severe stagnation and retention stages, the stomach, with its often weakened attachments, may become prolapsed. In obstructive stagnation and retention the same displacement may result. In complicated gastroptosis the contents of the stomach may stagnate or be retained. Consequently, the mere presence of gastroptosis does not prove that there may not be a primary myasthenia or obstruction. If there is no stagnation, myasthenia is thereby excluded, as is also a possible compensated obstruction, for the latter does not produce gastroptosis. If there is stagnation or retention, and if the physical signs of gastroptosis are present, the primary trouble may be the displacement, the myasthenia, or the obstruction.

Gastroptosis may produce a kink obstruction of the first part of the duodenum, and may with great difficulty be distinguished from other forms of obstructive stagnation and retention. In gastroptosis the reposition and support of the stomach remove the obstruction and cure the stagnation or retention, provided the muscular coat is not asthenic; this is an absolute differential sign. Rest in bed improves digestion in gastroptosis, immediately and greatly; it is much less beneficial in organic obstruction. The absence of any palpable cause of obstruction is in favor of gastroptosis. When the prolapsed stomach is myasthenic, the differential signs given in the chapter on Pyloric Obstruction become applicable.

Is the myasthenia or the gastroptosis primary? The history and the existence of associated displacements may throw some light on the case, but without a knowledge of the evolution, an etiological diagnosis is not possible.

Prognosis.—Gastroptosis is seldom completely cured—the displacement may be corrected, but it readily returns. A cure is possible in the early stages, particularly when a flabby abdominal wall is the occasion of the prolapse of the organ. However, by proper treatment great and often complete relief can be given in even its most advanced stages.

Treatment.—The treatment of gastroptosis is simple and methodical, and will usually prove efficient. The indications to be met may be thus formulated: (1) To replace and support the stomach; (2) to regulate abdominal tension; (3) to nourish the patient properly; (4) to prevent stagnation; (5) to treat the complication; (6) to relieve the symptoms.

Before replacing the stomach it is first necessary to make room for it. Whether it be forced out of position in the manner already described, or let fall by its relaxed supports, or dragged out of position by the attached colon, the room which it normally occupies is filled by other organs or by the collapse of the pliable wall of the upper abdominal cavity. Consequently, the spleen and liver, if enlarged, should be reduced in size by proper treatment; a compressing tumor or thoracic effusion should be removed. The constriction, collapse, and arrested development of the waist should be corrected by properly-fitting clothing, by the support of all skirts from the shoulders, and by methodical breathing exercises, combined with arm and trunk movements. The development and expansion of the upper abdominal wall can also be aided by massage.

The restoration of the normal space for the stomach, it need not be said, is much more difficult in some cases than in others. A stomach which has recently been dragged down into the flabby lower abdomen by the weight of its stagnant or retained contents or by the attached prolapsed colon is more readily replaced than one which has been forced out of position or one whose normal position has been filled by the hardened, collapsed, and badly developed costo-cartilaginous framework. The prolapsed stomach, like the contents of a hernia, may forfeit its right of domicile. When the gastroptosis occurs in young girls, and the expansion and development of the waist have been arrested by tight clothing, it is exceedingly difficult and often impossible, later in life, to recreate for the stomach its lost normal space. Natu-

rally, the stomach can not often be returned to its normal position like a reducible hernia. Gradual replacement of the stomach, however, is essential to a perfect cure; otherwise the treatment can only be palliative and arrest the further evolution of the trouble.

In order to support the replaced stomach the elasticity of its ligaments must be restored, the abdominal wall strengthened, and the intestines—which form a sort of cushion on which the stomach rests when the body is erect—be held well up in the abdomen. Massage, electricity, improved nutrition, and neuromuscular tone are slowly-working but valuable aids to this end. But to give immediate relief, the lower abdomen must be supported by an elastic belt.

The pelvic or hypogastric belts of Glénard, Teufel, Bardenheur, and Landau, when made so as to fit, and slightly modified so as to suit each individual case, do well the work which is required of them—an elastic and gentle compression and proper support of the contents of the abdominal cavity below the navel. We have found it best to have the belt made to order, and to adapt it, by modification, to the requirements of each case. The belt should be applied next to the skin in the morning while the stomach is empty, with the patient in bed; it should be tightened from below upward. In severe cases the bandage should be applied while the patient is in the knee-chest posture. When the abdomen is very flabby it may be necessary to wear the belt day and night; but usually, unless abdominal tension is very low, it may be removed before retiring.

In nearly every case of gastroptosis which has not been produced by downward pressure and by the excessive weight of the retained contents, intra-abdominal tension is too low. This is one of the causes of the inefficient peristalsis and of the stasis of the contents of the digestive tube. But persistent low tension means venous and particularly lymph stasis or congestion. This abdominal stagnation is pernicious in its influence on the nutrition of the abdominal and pelvic organs, and on digestion, absorption, and assimilation. Consequently, the coexistence of gastroptosis and low abdominal tension strongly condemns purgation or reducing treatment of any kind. Rest, massage, electricity, an elastic abdominal belt, a proper diet, supplemented, if need be, by gentle laxatives, are more rational and beneficial.

Rest in bed is a most valuable remedy. During the development of gastroptosis—and the displacement may often be detected in its beginning by a careful physical examination—

the patient should be given a methodical rest-cure. The evolution of the trouble would be at once cut short; the dragging pains, the tender points, nervousness, neurasthenia, and anemia grow less. Rest is essential in the cure of the secondary anemia and neurasthenia of gastroptosis. The mild cases of total descent of the stomach—and these cases are numerous—may be treated with only the amount of rest requisite to good digestive hygiene—half an hour's rest before and after each meal. Often it will be advantageous to enforce rest on the lounge during the greater part of gastric digestion.

The diet in gastroptosis is variable on account of the large number of complications, and is determined by the functional power of the stomach, by the state of the intestines, and by the needs of nutrition. As a rule, in gastroptosis the patient is pale, somewhat emaciated, and badly nourished. Abdominal tension is decreased and the organs are more easily displaced as the mesenteric, omental, and kidney fat disappear. At least a diet of support is an invariable rule, and it is best to improve nutrition as rapidly as possible by proper and nourishing food, such as each particular patient can utilize. A reducing and insufficient diet can only do harm in simple gastroptosis, but it may be made necessary by a severe complication, such as chronic colitis.

Gastroptosis, when associated with healthy intestines and good motor compensation, requires only a nourishing diet, measures to open the way gradually for the return of the stomach to its normal position, and good digestive hygiene. The diet should consist of plain and nutritious food, tender meats, fish, game, eggs, thoroughly cooked cereals, the more digestible vegetables, and a moderate quantity of sweets and fruits. The fat should not be diminished. In this stage there is no objection to milk, if it is digested by the particular patient. Pastry, rich sauces, coarse vegetables, indigestible articles, and most alcoholic drinks should be excluded.

Gastroptosis with myasthenia requires a diet suited to the stages of this complication; this diet has been given in the chapter on Myasthenia Gastrica. The drugs which have a special tonic action on the involuntary muscles should be prescribed—strychnin, hydrastinin, cinchona, and sometimes ergot and ipecac; all in small doses. Electricity and hydrotherapy are even more valuable. Faradism, both general and gastric, should be used, and the needle bath with hot, followed by cold, water over the abdomen and lower extremities. All hygienic measures which give tone to the

neuromuscular system should be employed. Briefly, the treatment is that of the myasthenia (already given) plus the treatment of the displacement. Rest in bed for a variable period is sometimes necessary.

Gastroptosis associated with chronic colitis requires a very carefully selected diet, abdominal support, digestive hygiene, often rest in bed, the cold or hot compress, the prevention of stasis, and the use of alkalies. The diet, for a short period, should consist almost exclusively of tender or chopped meats and poultry (white meat), either roasted or broiled, a little dry toast, crust of roll, the expressed juice of meats, once a day a few spoonfuls of a clear vegetable soup thickened with thoroughly cooked barley gruel, rice, or decorticated whole meal. No wine, milk, acids, fruits, or vegetables should be allowed. Half an ounce of fresh, unsalted butter should be eaten daily. During the employment of this restricted diet the patient should spend nearly the whole time in bed or on the lounge, in order to reduce the needs of nutrition to a minimum. After a few weeks fresh lean meats, preparations of wheat or rice, the white of eggs with a little of the yolk, the juice of a few grapes, a baked apple, stewed prunes, and the juice of an orange may be added. A few months later a mixed diet of plain foods should be prescribed, and rigorously adhered to until the patient is well. A glass of Vichy (Célestins, Grande-Grille) should be given daily, an hour before breakfast, particularly when the stools are very acid, contain much fat, or the urine deposits uric acid on standing; enough sulphate of soda should be added to the Vichy to produce a full and soft stool. The abdominal belt decreases the constipation, and the compress relieves the excessive irritability. If the motor power of the stomach is normal, a dose of Carlsbad salts or other saline (only one movement) may be given daily, or three times a week for one month. Cascara is the most harmless and valuable laxative. Hydrastinin, strychnin, cinchona, ipecac, and possibly belladonna, are often useful in small doses. But only the gentlest measures that are effective in the particular case should be employed to prevent the stasis of the contents of the colon. The localized spasmodic strictures of the colon are sometimes very much benefited by injections of warm pure sweet oil, to which two teaspoonfuls of oil of hyoscyamus (G. P.) may be added.

Gastroptosis with neurasthenia requires rest in bed. The digestive tube must be kept perfectly free from irritation by food, by fermentation or putrefaction products, and by drugs.

A milk- and rest-cure often yields excellent results when combined with massage, electricity, hydrotherapy, and proper moral treatment. If pure milk disagrees, it may be tried combined with alkalies, or a fermented preparation of milk may be substituted. Cod-liver oil may often be given at the same time with benefit. White meat of poultry and game, fresh lean fish, preparations of wheat and rice and other cereals, a little dry toast and butter, and sometimes eggs, may be added to the list after a few days. After all the neurasthenic signs are gone, red meats, green vegetables, and unirritating fruit may be permitted; but their digestion, utilization, and subjective effects should be watched. Most drugs are badly borne, and should be employed sparingly, and chiefly to meet functional indications. As in other forms of gastroptosis, the stomach should be supported and abdominal tension regulated by a suitable hypogastric belt.

It will thus be readily seen that the treatment of gastroptosis does not consist in the routine use of certain remedies. Each case requires special remedies, particularly when the disease is complicated. Each individual has his own peculiarities and constitution, demanding particular modifications of the medication employed. But all cases are alike in requiring prolonged and methodical treatment in order to establish and maintain digestive compensation or to effect a cure. Pain, insomnia, headache, nervousness, and uricemia often require symptomatic remedies.

CHAPTER V.

OBSTRUCTION OF THE ORIFICES.

The organic obstruction of the cardia and of the pylorus are diseases—or, more properly, deformities—which may require operation for their relief. The obstruction is nearly always due to ulcer, to cancer, or to toxic gastritis, and is then only an episode or a sequel of these diseases. But there are also other rare causes, and when the obstruction occurs a condition results which has a characteristic symptomatology, demands special treatment, and presents many difficulties in differential diagnosis. The practical value of a close study of this subject is obvious, for the surgeon stands hard by, ready to put the physician's opinion to the test.

1. **Obstruction of the Cardia.**—Obstruction of the cardia is a very serious condition, usually preventing the ingestion and absorption of enough food to maintain the nutrition of the body. The inanition is more or less complete and rapid, according to the degree of the stenosis. The body may sometimes be nourished on a carefully selected diet after it is no longer possible to swallow solid food. But the obstruction may become so great as to allow only a little or even no liquid nourishment to pass. The beginning of inanition, except in cancerous obstruction, marks a turning-point in the treatment of the condition; and then feeding by bowel, through an esophageal cannula or through a gastric fistula, must be employed. Surgical interference is imperative when starvation begins.

Etiology.—Obstruction of the cardia is much less frequent than the same deformity of the pylorus, but both are alike in the multiplicity of their manner of production.

The causative disease originates in the wall of the digestive tube in the vicinity of the orifice, or the obstruction may be due to the compression of a mediastinal or retroperitoneal tumor, a cold abscess, enlarged glands, or aneurysm. Congenital atresia is exceedingly rare, the ectodermic esophagus developing and forming a complete and perfect union with the endodermic stomach. Congenital obstructive malformation of this part of the digestive tube is scarcely known.

Stenosis of the cardia may be spasmodic, and is then commonly known as esophagismus. Spasmodic stricture is not very rare, and may occur at any age. Organic strictures are often made tighter by spasm of the cardia.

Cicatricial contraction is one of the common causes of organic obstruction. Tubercular and syphilitic ulcers very seldom occur at this point of the digestive tube. Ulcer of the stomach does not often involve the cardia. Catarrhal and corrosive ulceration is more frequent, the cardia being the first point of arrest in the gullet of corrosive acids, alkalies, or salts. Consequently, these chemicals commonly produce stricture of the lower part of the esophagus.

The most common cause of cardiac stenosis is cancer, extending to it either from the esophagus or the stomach, and composed histologically of pavement or cylindrical cells. If it be remembered that the number of deaths due to cancer of the stomach is small, and that only a small number of gastric carcinomata involve the cardia, it will readily appear that stenosis of the cardia, including all its varieties, is not a frequent deformity.

Clinical Description.—The manifestations and evolution of obstruction of the cardia are variable. Some of the symptoms are due to the obstruction itself and to its consequences—dysphagia, esophageal stagnation, and inanition. Others are due to the causative disease located in the wall of the tube or in its vicinity. The modifications of the clinical picture entailed by the method of production will be discussed under the etiological diagnosis.

The first symptom of obstruction is the arrest of food in the lower portion of the esophagus. The patient states that solid food seems to stick at that point, and that it requires a deep inspiration and repeated efforts at swallowing to force it on. This sensation will most likely be noticed first after a large bolus is swallowed, but later small mouthfuls of solid food and the very rapid gulping of liquids produce the same sensations. Solid food can be made to pass readily only by swallowing a little at a time and washing it down with fluids. The meal can be finished comfortably only when eaten slowly and in small mouthfuls. The obstruction manifests itself only during eating.

Soon the signs of esophageal stagnation appear. After the meal is finished there seems to be, beneath the lower end of the sternum, a foreign body which gives a good deal of discomfort and anxiety from the compression which it exerts. The symptom is in direct relation with the solidity of the food, the rapidity of eating, and the heartiness of the meal. The food frequently regurgitates into the mouth, particularly after coughing. Chemical tests will show that the matter regurgitated has not entered the stomach, but it is often mixed with a good deal of mucus from the local irritation which the stagnation or the causative disease excites. This sensation of a foreign body beneath the sternum may be due in part to the obstructing tumor.

Later, stagnation is replaced by retention. The food ferments, decomposes, irritates, and the contents of the dilated pouch are regurgitated again and again into the mouth, mixed with mucus and saliva; they are sometimes foul, and contain no bile, hydrochloric acid, pepsin, labferment, nor products of gastric digestion.

The stagnation and retention produce secondary thoracic symptoms. There may be dyspnea, often discomfort or pain, increased by exercise, and sometimes attacks of pseudo-angina pectoris. The obstruction may be increased by spasm of the cardia and by inflammatory swelling, or it may be diminished by ulceration. Consequently, organic obstruction

of the cardia usually presents periods of diminution and of increase, and eventually, and more or less rapidly, in keeping with the nature of the cause, may become complete.

If, on account of the obstruction, too little food is received into the stomach, nutrition suffers. The symptoms are those of simple subnutrition—thirst, hunger, emaciation, weakness, and the particular functional and nervous signs due to a constant deficiency of food and water. The stomach and bowels are particularly liable to retract. To the obstructive and denutritive manifestations will naturally be added those expressive of the causative disease; for the obstruction of the cardia itself may be congenital, or an episode, or a sequel.

Symptomatology.—One of the earliest signs of obstruction of the cardia is the arrest of solid food in the lower portion of the esophagus. The food sticks at this point and requires repeated efforts to force it on. In the case of ulcer, this arrest is preceded by a period when the swallowing and the movements of active respiration are painful. As the ulcer heals and the cicatrix forms, the pain gives place to obstruction. First solids, and then liquids, are arrested, and, finally, only a small quantity of liquid food or none can be forced into the stomach. Cicatricial constriction often stops before occlusion is complete, and remains for a long time stationary. The obstructions of carcinoma and of growing and compressing tumors are progressive. In cancer the obstruction may be diminished by ulceration, and the consequent improvement in swallowing gives rise to false hopes. The obstruction is often increased by spasm and swelling, and the food passes more easily when the irritation and inflammation subside. The irritation produced by passing a sound may cause complete occlusion for a few days. The period of compensatory hypertrophy of the esophagus is short when carcinoma is present, and inspiratory compression, instead of forcing the food into the stomach, only brings it up into the mouth. In organic obstruction there are no periods when swallowing is effortless and normal, as there are in esophagismus.

The regurgitation is at first alimentary, and is limited to the period during the taking of food or shortly afterward. Later, as stagnation and retention develop, it occurs between meals, consisting of repeated mouthfuls of food, of saliva and mucus, or of all mixed together, and being sometimes fermented and putrid, particularly in carcinoma. The persistent absence of any chemical evidence that the regurgitated matter has entered the stomach is characteristic of esophageal

vomiting, which in some cases may be accompanied by nausea. Blood may sometimes be brought up when there is ulceration or when there are dilated cardio-esophageal veins.

Pain may be entirely absent or it may be severe, and where there is no great stagnation or retention it may be confined to the mealtime. Sometimes, however, as the pocket forms and fills, or when there is ulceration, the pain is severe, located near the ensiform process, and radiates into the back and concentrates about the heart. It may be accompanied by dyspnea or hiccup, and is likely to be increased by exercise and deep breathing. Sometimes the pain is excited by walking, and disappears as the esophagus empties itself.

The physical signs are very valuable and characteristic. A tumor of the cardia can neither be felt nor seen. Sometimes the filled esophageal pouch produces dulness posteriorly to the left of the eighth or ninth dorsal vertebra. Depression and percussion over the lower end of the sternum are sometimes painful; seldom a painful pressure-point exists posteriorly and to the left of the ninth dorsal vertebra. These are, however, only suggestive and not revealing signs. When the stenosis is so great as to prevent the ingestion of enough food, emaciation and loss of strength develop. If water also is more or less excluded, the inanition becomes more marked, and the wasting of the fat and muscles may become extreme; there may be great prostration, sunken abdomen, contracted stomach and intestines, and the general skeleton-like appearance of food-and-water starvation. The quantity of urine passed daily is a fair measure of the degree of obstructive retention.

The swallowing sounds may be of some diagnostic value. There is not likely to be a stenosis when, after a swallow of water, the first and second sounds are both heard, separated by an interval of about twelve seconds. If obstruction is present, both sounds may be absent, or the appearance of the second sound, which marks the completion of the passage of the water swallowed, may be delayed, or, if the esophagus contracts with little force, the second sound may be inaudible. These auscultation signs and the other foregoing physical signs are only suggestive or presumptive.

The characteristic physical sign is obtained by sounding the esophagus. This should first be attempted with the stomach-tube, using various sizes. For this purpose a tube with a single side-eye near the extremity is preferable, inasmuch as it makes it possible to locate and to measure the length of the stenosis more exactly. If the eye be above

the stricture, only a little water can be introduced, and this can be removed by siphonage. If the eye be beyond the obstruction, the water passes into the stomach. If the eye is within the stomach, the introduced water can be removed by expression. The stricture of the cardia thus located and its length measured (provided it can be passed), is about 40 cm. from the point where the incisor teeth touch the introduced sound. The solid, flexible, black-rubber esophageal sounds may also be employed to detect and measure the distance of the stricture from the incisor teeth. The olive-pointed bougies are dangerous.

A piece of the tumor may be sometimes withdrawn in the eye of the stomach-tube. Blood is more likely to be obtained when the obstruction is due to cancer, and the substances withdrawn on or in the tube in malignant disease are sometimes of a foul odor. These anatomical signs, which reveal the malignant nature of the obstruction, are obtained frequently enough to attract and deserve attention.

Diagnosis.—The diagnosis is easy if the clinical history be sufficiently clear to suggest the use of the sound, and if the latter be found to meet with resistance about 40 cm. from the incisor teeth. The slight normal resistance of the cardia to the passage of the sound will produce no suspicion in the mind of one with some experience in the use of this instrument. Other symptoms and signs may suggest the situation of the trouble. But, unfortunately, in the early stage the sensations of the patient, and particularly his description of them, are somewhat vague. The patient may only complain of a little shortness of breath, fullness and oppression in the chest, a slight irregularity or rapidity of the heart's action, and pain, which is particularly excited by exercise, and which disappears after a few moments in spite of the continuance of the exercise. The layman is most likely to be anxious about his heart, and to have this organ in his mind, often denying any difficulty in swallowing, as there is no pharyngeal trouble. "I fear my heart is diseased; I care nothing about the regurgitations and other dyspeptic symptoms," is a statement somewhat frequently made in this early stage. A history of this kind should direct the attention of the physician to the cardia.

After the certainty of the existence of an obstruction of the cardia is established, the detection of its cause is practically very important, and sometimes difficult. Particular attention should be given to the evolution and persistence of the symptoms and to the character of the signs. Is the stricture organic, and, if so, what is its nature?

Spasmodic stricture of the cardia occurs chiefly in neuropaths. It is most common in neurasthenic or hysterical girls, about the age of puberty, and in women during the menopause. Men, particularly of the arthritic types, are by no means exempt. But a favorable temperament or constitution can only excite suspicion of the spasmodic nature of the stricture. There are, however, three cardinal and pathognomonic signs: the obstruction is intermittent; a large sound passes as readily as a small one after both have been arrested (gentle and sustained pressure is often sufficient to overcome the resistance); and antispasmodics relieve the obstruction. If the stomach-tube does not pass after gentle and prolonged pressure, and if no anatomical signs are obtained upon its withdrawal, the patient should be quickly put under the influence of the bromids. The spasm will then relax, and no obstacle will be encountered in passing the tube. To avoid repetition, the reader is referred for other distinctive signs to the article on Spasm of the Cardia.

The cause of the organic stricture is determined by exclusion. The age of the patient, the duration of the trouble, and the signs of a disease likely to cause obstruction, all have a differential value. If a corrosive poison has been swallowed, or if the esophagus has been burned by hot food or water, or if there have been symptoms and signs of an ulcer, the stenosis is most probably cicatricial. In the absence of such a history, the obstruction is most probably cancerous, particularly if there are other constitutional and local signs of malignant disease and if no disease which is likely to compress the digestive tube at this point can be discovered.

Prognosis.—The prognosis is dependent on the nature and degree of the stenosis. The auto-intoxication and inanition of carcinoma prove fatal in five or six months. Cicatricial stenosis, after the scar tissue has finished contracting, and when the stricture is of large caliber, may permit the maintenance of nutrition by careful alimentation. Even a cicatricial stricture of small caliber leaves room for some hope of relief by surgical treatment. The prognosis is never good and should always be guarded.

Treatment.—The treatment consists in appropriate alimentation, so as to maintain, as long as possible, the balance of nutrition, the control of fermentation and putrefaction when they exist, the protection of the diseased part against injurious irritation, and the employment of surgical procedures to increase the caliber of the stricture, to maintain its per-

meability, or to form an artificial gastric fistula through which the patient can be fed.

The two important qualities of the food are its nutritive value and its power to pass the obstruction. The caliber of the stricture has the most to do with the selection of the diet. So long as there is present an active inflammation or excessive irritability, the food should be warm, fluid, and unirritating, so as not to increase the swelling nor to excite spasm. The artificially digested foods possess no advantages over the fresh fluid preparations. Milk, raw eggs, expressed meat juice, the juice of the grape and pineapple, and of other unirritating and nutritious fruits, meat powder, cocoa, chocolate, and similar fluid preparations suitable to the digestive power and nutritive state of the individual patient should be ordered. Nutrient enemata should be employed as soon as alimentation by the mouth is insufficient.

When stagnation and retention are present, fermentation and putrefaction may be distressing and injurious. The mouth, nose, and throat should be kept as sweet as possible. A little brandy may be given from time to time with advantage, and the least irritating antiseptics may be tried. If the esophageal pocket is large and retention is present, the contents should be withdrawn before fermentation becomes active, and the cavity should be washed out with warm Thiersch's solution. Methylene-blue may be used when the stricture is cancerous, or the iodid of sodium and arsenic may be prescribed (Boas). Codein may be required to relieve pain or spasm of the cardia.

The surgical procedures are numerous. In cicatricial stenosis gradual dilatation with flexible rubber esophageal sounds or more rapid dilatation with the balloon catheter may be undertaken. This treatment may be employed early, while the stricture is of large caliber, and should be conducted on the same principles as the gradual dilatation of a urethral stricture. If the cicatricial stenosis is impermeable, or if its caliber is so small as to prevent the ingestion of sufficient food, gastrostomy may be performed and an effort may be made to dilate the stricture from the stomach, with a view to continuing the dilatation later by the mouth. In the meantime the patient should be fed through the fistulous opening. Sometimes the stomach is so small and lies so deep that gastrostomy is impracticable. Jejunostomy should then be performed instead.

In cancerous obstruction gradual dilatation can serve no purpose. When the quantity of food introduced by mouth

and rectum is so small as to entail rapid emaciation and loss of strength, gastrostomy may be performed, but with little hope of prolonging life or of diminishing the discomfort.

2. **Obstruction of the Pylorus.**—Pyloric obstruction is usually described among the causes of "dilatation" of the stomach. It is true that pyloric obstruction may eventually produce enlargement of the stomach; but the disease should not be named after one of its final effects, for practically and essentially the trouble is the obstruction. The word "obstruction," however, does not cover the whole period of the genesis and evolution, for before the beginning of the obstruction there may have been some inflammatory or ulcerative process or some local nutritive or developmental trouble. But whether the obstruction be a complication or a sequel, congenital or neoplastic, a new danger is added, and the clinical picture and treatment are changed by it. It becomes practically a morbid process, enforcing changes and consequences manifested by a group of symptoms. Nor does the disease begin, as is often tacitly assumed, with "dilatation." The final period of gastric retention is preceded by periods of compensation and of stagnation.

Neither is pyloric obstruction synonymous with obstruction to the evacuation of the stomach, for stagnation and retention are frequently caused by duodenal obstruction. These two symptoms (stagnation and retention) may be produced by myasthenia, by supersecretion, by pyloric obstruction, and by duodenal obstruction. Pyloric obstruction is a result or accompaniment of a number of distinct diseases of the stomach, which are of the very greatest medical and surgical interest. Its forms should be differentiated and carefully studied, with a view to their recognition when met with at the bedside.

Etiology.—Pyloric obstruction may be either organic or due to spasm of the pyloric muscle. The spasmodic form is described in Section iv.

Congenital atresia of the pylorus is very rare, but is more common than the same defect of the cardia. The digestive tube may be represented at this point by a fibrous cord, or, more frequently, the canal is not completely obliterated, but is of small caliber. The development of the pylorus may be arrested at any stage, and the infant may die of inanition a short period after birth or may grow into manhood by observance of a suitable diet.

Another cause of pyloric obstruction is cellulomuscular hypertrophy and hyperplasia. The increase in size may be so great as to present a veritable tumor, consisting of a large number of circular muscular fibers and increased submucous connective tissue. This benign hypertrophy of the pylorus is of two distinct varieties—simple and inflammatory. The simple hypertrophy is of a purely functional or nutritive nature. The inflammatory hypertrophy or the hyperplasia is a sequel of chronic hypersthenic gastritis, the lumen being made smaller by hyperplastic thickening of the pyloric wall. The productive inflammation and hyperplasia involve the mucosa, the submucosa, and the muscular layer.

Very seldom the pylorus is obstructed by a foreign body. This may be produced by a wandering gall-stone, a gastrolith, or a swallowed foreign body which has obtained lodgment in the pylorus.

Obstruction by a benign tumor is also very rare. The obstructing tumor may develop in the gastric wall close to the pylorus, or the canal may be closed by a polyp becoming engaged in it.

Syphilitic and tubercular ulceration is scarcely known at this point of the digestive tube. More frequently the pylorus is obstructed by the contraction of the scar tissue resulting from the destructive action of chemical poisons.

The pylorus is sometimes compressed by a tumor developing in its vicinity, or by a gall-stone in the common bile duct, or by constricting bands of fibrous tissue resulting from peritonitis, or by displacements of the stomach. Obstruction of the duodenum, however, is more frequently thus produced.

Ulcer is one of the common causes of pyloric obstruction. During the evolution of the ulcer the obstruction may be due to the inflammatory swelling and thickening of the walls of the ulcer or to pyloric spasm. More frequently the obstruction is due to the deformity produced by contraction of the ulcer scar.

The most common cause of pyloric obstruction is undoubtedly cancer. Here the location and direction of the growth of the neoplasm are more important than its size. The carcinoma is very often a small annular scirrhus.

Clinical Description.—Pyloric obstruction presents three stages—the periods of compensation, of stagnation, and of retention. The duration of these periods is dependent on the nature and rapidity of the development of the obstruction and the readiness with which the muscular layer of the

stomach undergoes compensatory hypertrophy, which may more than double the thickness of the stomach wall. The hypertrophic thickening is always greatest near the pylorus.

The period of compensation is characterized clinically by the very great disturbances produced by dietetic excesses, particularly the eating of large quantities of coarse food. The muscular hypertrophy compensates only on condition that the stomach is not given too much mechanical work to do. A small meal that is finely subdivided by proper preparation and thorough mastication will be comfortably digested. Even a large meal of the same character may be digested and evacuated by the stomach without perceptible trouble. Fluids are more readily evacuated than solids. During the active period of gastric digestion the stomach can be felt periodically contracting. All these characteristics are absent in myasthenia, with which pyloric obstruction is often confounded. But a large meal composed of coarse, solid food, made excitant by condiments, produces stormy peristalsis, colic, and often vomiting. The vomit is alimentary, often contains but little fluid, and is frequently excessively acid, not from fermentation, but from secretory irritation. The active churning movements, and the intermittent and somewhat accidental damming of the current, are unfavorable to germ growth. The period of compensation, marked clinically by recurring attacks of motor disturbance produced in a particular manner, may be long or very short. In benign hypertrophy of the pylorus, in congenital stenosis of large caliber, and in moderate cicatricial obstruction, compensation may continue for years. Pyloric spasm may play an important part in the development of the attacks, particularly when an inflamed ulcer is located near the pylorus. The period is usually very short in obstructing carcinoma, in cases of ulcer which rapidly close the canal, and in severe cases of ulceration or of corrosive toxic gastritis.

The period of stagnation may mark either the beginning of the obstruction or the beginning of failure of compensation. The stomach empties itself slowly, and never contains in the morning before breakfast, as it does in retention, food which was eaten the evening before. Delay in the evacuation of the stomach, which is one of the signs of functional stagnation, may be but slight, or, in the severe cases, the stomach may be found empty at only one period during the twenty-four hours, this being in the early morning, during the long interval between the evening meal and breakfast. The stomach trouble may manifest itself, in the same

manner as in the first stage, only during accidental failure of compensation; or the stagnation may produce symptoms after each meal. The irritation may not be sufficient to cause much disturbance after breakfast, but when the stomach does not empty itself between breakfast and the evening meal, there may result an accumulative effect manifesting itself each afternoon or evening. The irritation of the fermenting contents and the excessively acid secretion may produce, particularly in the cicatricial stenosis of ulcer, intolerance of food by the stomach and a rebellious form of alimentary vomiting. The vomit contains food mixed with a clear, acid fluid holding a large quantity of albumoses in solution. When no compensatory hypertrophy has developed there may result an asthenic state, with a sensation of weight, with belching, and with acid regurgitations, particularly in carcinoma. Pain, and sometimes very severe pain, is likely to be a symptom of the stagnation. Emaciation, but often very slight, occurs even in the mild cases; and when the stagnation leads to gastric intolerance, inanition develops rapidly. This stage, also, according to the rapidity of the evolution of the causative disease may be either long or short.

Retention, or the third period, is characterized by the failure of the stomach to evacuate its contents at any time during the twenty-four hours, by copious vomiting of accumulated foods and large quantities of fluid and digestive products, and by more active fermentation with gas formation. Pain, as in stagnation, not only encroaches on the period of normal gastric repose, but also often continues throughout that period. The symptoms due to the absorption of an insufficient quantity of nutriment and water are combined with those of gastric auto-intoxication. Emaciation and loss of strength may be very great, and the patient may become markedly cachectic. This stage may result from the obstruction becoming greater and greater or from the failure of the muscular layer of the stomach to maintain compensation.

Congenital atresia proves rapidly fatal. The infant may be well enough at birth, but uncontrollable vomiting soon begins, and death follows without any food or drugs ever having passed through the alimentary canal.

Congenital stenosis produces symptoms which depend upon the caliber of the stricture. If the canal be very small, the stomach, after enlarging, and without undergoing hypertrophy, becomes intolerant, and death results in a few months from inanition. If the caliber be larger, the stomach increases in size and strength, and compensation may be established.

At no time is there a palpable pyloric tumor, and the symptoms of obstruction begin soon after birth. These children with large and hypertrophied stomachs are in constant danger of the failure of compensation and the development of retention. The obstruction becomes uncompensated during each attack of a severe acute disease, and adds largely to the danger.

Simple benign hypertrophy of the pylorus is produced in two ways. The one is functional and nutritive, and develops and produces obstruction in the same manner that the strong and irritable anal sphincter causes constipation. The other is due to duodenal stenosis, the pyloric ring developing so as to aid in establishing compensation and prevent the reflux of the contents of the duodenum into the stomach. In the first form gastric compensation may be complete. In the second, compensatory hypertrophy of the muscular layer of the stomach is maintained with greater difficulty, and the stomach may often contain bile and pancreatic juice, the expressed contents after being rendered alkaline, digesting albumin, and also emulsifying oil. Benign hypertrophy may present in its evolution the three stages of compensation, stagnation, and retention. The disease usually develops slowly, and often lasts a number of years.

The clinical expression varies little, whether the hypertrophy be functional and nutritive or the result of hypersthenic gastritis. The three clinical periods are well marked, and the duration of the disease, which is frequently fatal, is long. During the first period the accidental disturbances of compensation produce the morbid features, the intervals between them being symptomless. These recurring attacks, produced by dietetic excesses or errors, may be short or may last one or two weeks. Three or four hours after the excessive meal gastric pain commences, increases in severity, and is greatly relieved by the vomiting which ensues, the characteristics of which demonstrate the stagnation of the food in the stomach. The vomit contains food, often a good deal more fluid than was swallowed, and is excessively rich in hydrochloric acid and mucus. After the vomiting the pain subsides, and the following meal may be evacuated properly, demonstrating the reëstablishment of compensation. The attacks, however, may last a week or two, and during this period the pain varies in intensity, often completely subsiding for a few hours. The vomiting occurs soon after a meal of solid or liquid food, or later, at the time when the stomach should normally be empty. The bowels are obstinately con-

stipated. These attacks may recur after an intermission of weeks, or even months.

During the period of severe stagnation the manifestations are more continuous, and pain is likely to recur after each meal, in relation with the evolution of secretion and the activity of peristalsis. The gastritis is increased by the irritation of the acid contents, which remain abnormally long in the stomach. The stomach may become intolerant, or vomiting may occur only once or twice in the twenty-four hours. Constipation is obstinate. The pylorus is exceedingly sensitive, and both anteriorly and posteriorly pain upon pressure is marked, and extends over a large area.

When retention begins, the clinical picture changes. The vomiting is copious and, usually, less frequent. The pain is very severe, and may continue day and night. Liquids as well as solids are retained too long in the stomach. The vomit is liquid, separates into three layers, and contains products of hydrochloric-pepsin-digestion, and organic acids. Gas-forming fermentation is active in the tube-tests. The bowels are obstinately constipated, and the quantity of urine is small, constituting a rough measure of the degree of stenosis. The appetite is now lost and the emaciation may become cachectic. Death follows from inanition and auto-intoxication.

Obstruction by benign tumors is exceedingly rare, and presents, like syphilitic and tubercular ulceration, no distinctive clinical expression. Toxic gastritis more frequently produces obstruction of the cardia than of the pylorus. The development of the usual signs of pyloric obstruction after the swallowing of a corrosive chemical reveals the character of the trouble. The stenosis may be of large caliber or may produce complete occlusion. The strictures of large caliber present a period of stagnation or retention, and may become compensated by gastric hypertrophy after a few weeks. The compensatory hypertrophy produces the only improvement, cicatricial stenosis being permanent and the contraction of the scar tissue being often rapid.

The stenosis of ulcer may occur during the evolution of the ulcer, or during or after its healing. If it occur from inflammatory swelling during the period when the ulcer is still progressive, gastric intolerance is rapidly developed. The vomiting usually occurs in from one-half to two or three hours after the meal, and often contains food eaten eight or ten hours previously. The development of obstruction changes the clinical expression of ulcer—the pain increases,

vomiting becomes more obstinate, emaciation is rapid, and, with the stagnation and retention, fermentation also sets in, and is in no manner prevented by the excessively acid secretion. The obstruction may begin as the ulcer commences to heal, and its development is then commonly preceded by a period of improvement. The cardinal symptoms of ulcer, with the exception of hemorrhage, increase, those of obstruction develop, and the hypersthenic gastritis becomes more active. But more frequently obstruction develops after the ulcer has healed. Recovery has been marked by the cessation of all the symptoms of ulcer, the scar tissue is contracting, but the patient again becomes ill, and the signs are those of obstruction combined with hypersthenic gastritis. The cicatricial stenosis of ulcer may present the three periods of compensation, stagnation, and retention; and the keynote of the clinical expression is persistent and often violent pain.

Cancerous obstruction is the most frequent form. It often develops with great rapidity, and marks a turning-point in the clinical history of carcinoma. Stagnation and retention may occur in cancer from the loss of functional activity of the muscular layer. But when obstruction begins, the denutrition becomes more active, vomiting is likely to be more obstinate, and the fermentation generates large quantities of gas, both in the stomach and in the fermentation tube tests.

However caused, pyloric obstruction presents three degrees or stages—compensation, stagnation, and retention; but the clinical expression varies in relation with the nature of the underlying cause. In carcinomatous obstruction even temporary compensation is exceedingly rare.

Symptomatology.—The modifications of the appetite are dependent on many features of the disease. In the later stages the state of nutrition is of great influence. The causative disease exerts its modifying power. Anorexia is common in carcinoma. The appetite is, on the other hand, well preserved or even increased in benign hypertrophy, in ulcer, and in hypersthenic gastritis, the latter being sometimes a cause and sometimes a sequel of the pyloric obstruction. Mental and moral depression and nervous weakness, which are sometimes manifestations of pyloric obstruction, decrease the appetite; but apart from the state of nutrition and of the nervous system, apart from the nature of the causative disease, and in the absence of trouble in any other organ, pyloric obstruction modifies the appetite by the gastric fermentation, and the intestinal putrefaction, resulting from the stagnation

and retention. The appetite diminishes as the uncleanliness of the stomach increases. During the period of compensation the appetite is good and there is no excessive thirst. In stagnation the appetite is variable. But where pronounced retention exists, the appetite is likely to be poor, and thirst may be unquenchable.

Pain is one of the most common local symptoms, and is due to a variety of circumstances. It may be due to ulcer, to the associated hypersthenic gastritis and excessive secretion, or to fermentation. This form of pain is produced by irritation, and may be made severer by the irritable weakness of the nervous system. Another cause of pain is the peristaltic effort to overcome the obstruction, which is particularly marked during the stages of compensation and stagnation. The pain of retention is due also to irritation. The little accidents which disturb the period of compensation occur during gastric digestion, the peristaltic effort increasing and becoming painful during the height of digestion and subsiding after the evacuation of the stomach. During the period of stagnation the pain is both peristaltic and irritative, and may occur after each meal or only after the heartiest meal of the day. It ceases with the evacuation of the stomach. When retention begins, the pain is often paroxysmal, recurring once or twice a day or every few days. In other cases, particularly when there is excessive secretion, the pain hardly ceases day or night, and sometimes is intolerable.

Vomiting is almost as frequent as gastric pain. During the period of compensation it occurs as a terminal sign of the accidental or temporary muscular insufficiency. It then commonly occurs at the moment when the contents of the stomach should already have been evacuated completely into the duodenum. The peristaltic movements become more and more stormy, and are usually painful until the cardiac orifice is forced and the stomach is emptied by vomiting. This form of vomiting is very rare in myasthenia, but may occur in the muscular insufficiency due to an excessive meal or to nerve or muscle fatigue. The vomit which results from a temporary disturbance of compensation is alimentary and contains but little fluid. During the period of stagnation vomiting may occur a little later than the time when the stomach should normally be empty, either after each meal or, commonly, only once or twice a day, and as the final expression of an attack of peristaltic pain. This is the period when vomiting is most frequent and most obstinate. The stomach often becomes for a few days completely intol-

erant, and both fluids and solids introduced into it are almost immediately rejected. The stomach is completely emptied. Nausea is common during and after these attacks of intolerance, and may be associated with an almost neutral or excessively acid gastric juice. Secretion is likely to be very acid during the period of intolerance when an attempt is made to feed by the mouth, and there is then a good deal of pain and nausea. But after the force of the attack is spent, and even while the stomach is given functional rest, nausea is prominent, and is usually accompanied by a nearly neutral secretion and by occasional vomiting. The vomiting, except during the periods of intolerance, is not immediate, but occurs only a long time after meals. The vomit is alimentary, stagnant, and much more fluid than in the stage of compensation. The period of stagnation is the period of excessive formation of organic acids, and butyric acid is often the cause of the intolerance. During the period of retention the stomach may be intolerant, but only when the muscular layer is hypertrophied and irritable. When myasthenia develops, the stomach yields to distention without a struggle, and the vomiting becomes more copious, more fluid, and more infrequent. Once every few days vomiting occurs, and is usually effortless, painless, and incomplete. The stomach does not completely empty itself. This is the period when the fermentation tube tests yield large quantities of gas in addition to organic acids. The vomit is that of retention—overfermented, acid, fluid, and separating on standing into three layers. Many constitutional symptoms and distant effects have been attributed to gastric auto-intoxication. There is no organ of the body, it is said, which may not be affected by toxic products absorbed from the "dilated" stomach. (This theory is discussed in the chapter on Myasthenia Gastrica and in the section on the Vicious Circles of the Stomach.) Many of these accidents, particularly those which involve the nervous system, are seldom observed in pyloric obstruction not complicated by intestinal putrefaction. The contents of the stomach are neither absorbed nor evacuated, to a noteworthy extent, into the intestines, but are periodically removed by vomiting. The obstruction protects both the intestines and the system. But in myasthenic retention the gateway to self-poisoning remains open. Obstructive retention seldom produces melancholia, hypochondriasis, delirium, hallucinations of sight, diplopia, hemianopia, formication, cramps in the extremities, dyspnea, tachycardia, arrhythmia, irregularities of the pulse, acne, urticaria, ery-

thema fugax, and other disorders of the nervous, vascular, respiratory, and cutaneous systems, by means of toxic products absorbed from the stomach. These symptoms are more frequently due to intestinal toxemia, but indirectly — by mechanical compression, by reflex action, and by inanition— obstructive retention may exert a very pernicious influence on the various functions. The enlarged and distended stomach, filled with gas and acid contents, may compress the heart and limit the movements of the diaphragm. If the fermentation is butyric, acne is common enough, and vasomotor disorders of the skin may likewise be produced by the reflexes starting from the irritated gastric mucous membrane. Many nervous and cardiorespiratory symptoms may be induced by the secondary neurasthenia and anemia. Few of the constitutional symptoms are toxic, but some undoubtedly result from the inanition. The skin becomes dry and often scaly, the extremities cold, and the body a more easy prey to its environment. There is less resistance to bacterial invasion and diminished endurance of cold weather and of sudden atmospheric changes. The voluntary and involuntary muscles become weak, and tonic and painful cramps of the muscles of the forearm and of the calves of the legs are not rare. But these symptoms, with emaciation and loss of strength, constipation, and diminution of urine, are the result of inanition. The food and water are retained, or are lost largely by vomiting. Water may be eliminated in large quantities by excessive gastric secretion. The food, also, instead of being digested, ferments and decomposes. The result is a form of starvation accompanied by dry cachexia.

The nutritive state varies in the different stages of obstruction, and is influenced, also, by the nature and evolution of the causative disease. In the cicatricial stenosis of ulcer and of toxic gastritis the patient may, in the beginning of the obstruction, be emaciated and anemic. Cancer also produces albuminous denutrition before the pyloric canal is obstructed by the new growth. But, apart from the causative disease, pyloric obstruction affects nutrition in proportion to its degree. During the period of compensation, on a suitable diet provided it be supporting, there is no loss of weight or of strength. But when compensation fails and stagnation results, nutrition begins to suffer. The loss of weight and strength, however, during this period is due more to accidents, particularly fermentation and vomiting, than to the obstruction, for the stomach eventually empties its contents

into the intestines. When retention begins, nutrition fails more rapidly, and the body is not only insufficiently nourished, but excessively dry. When the retention is pronounced, the patient is emaciated and the skin is rough and dry. The strength, however, often does not fail so rapidly as might be expected. But when the obstruction is cancerous, or when intestinal auto-intoxication exists as a complication, the muscular weakness develops rapidly. Suddenly developing obstruction produces more rapid and complete starvation than results from any other disease of the stomach.

The local physical signs are of great diagnostic value. During compensation there may be no sign, unless a tumor of the pylorus can be felt. But after meals the region of the normal stomach is somewhat prominent; the stomach is more resistant to the fingers, and its form is well preserved, particularly during digestion; its walls feel elastic and firmly contracted, and the organ may be enlarged. If the abdominal wall is thin and yielding, even in this stage the strong peristaltic waves are sometimes visible, and the alternate relaxation and contraction of the stomach are often palpable at the height of digestion. During the period of stagnation the peristalsis, provided the stomach is not myasthenic, is strong and active, and is more likely to be visible, and gastric splashing may be elicited at the time when the stomach is inactive. But visible peristalsis can not be excited in the empty stomach in the morning before breakfast. During the third stage, or that of retention, two physical conditions may exist. The stomach may be strong and feel firm to the palpating finger, and the splashing sounds may be elicited with greater difficulty during peristaltic contraction than during the relaxation of the organ. The alternate relaxation and contraction may also be easily felt; or, on the other hand, the stomach may be flabby and easily compressible during its contraction, and the splashing sounds may be produced at every moment with equal ease. In both conditions gastric splashing is absent at no time during the twenty-four hours, and the abdominal wall over the stomach is prominent, except when the organ is emptied by the tube or by vomiting, and becomes more and more prominent as the stomach fills by accumulation. The comparatively empty and contracted intestines usually occupy but little space, and the stomach is, as a rule, increased in size, and contains a large quantity of gas when, physiologically, it should be retracted and empty.

A palpable tumor of the pylorus is a valuable sign of pyloric obstruction. Such a tumor may be formed by a neo-

plasm, by an ulcer with infiltrated walls, or by simple or inflammatory hypertrophy. To detect the tumor, the examination should be made when the stomach is empty and with the patient on his back. When the tumor lies beneath the false ribs it may sometimes be caught by the fingers at the end of a deep inspiration, particularly if the patient lies on the left side. Sometimes the tumor can be felt only when the stomach is moderately filled with gas, and, consequently, inflation may aid in locating the tumor. The pyloric tumor can sometimes be felt relaxing and contracting, and gas can be felt and heard bubbling through it synchronously with the peristaltic contraction of the stomach. But the absence of a tumor, even when the pylorus can be felt, does not exclude pyloric obstruction from arrested development and cicatricial stenosis. A small annular scirrhus produces little increase in the size of the pylorus, and the obstructing tumor may be out of reach. Frequently enough, neither the pylorus nor a pyloric tumor can be felt.

The functional signs vary according to the nature and degree of obstruction and of muscular insufficiency, the character of the contents, and the form, extent, and degree of the inflammation of the mucous membrane. (The functional signs of cancer, of ulcer, and of hypersthenic and asthenic gastritis are described in the chapters on these diseases.) Only the functional signs due solely to the obstruction need here receive attention. During the stage of compensation the obstruction causes no abnormalities in the digestion and evacuation of the test-breakfast and the test-dinner. Only when coarse, solid food is given is the evacuation of the stomach delayed. During the stage of stagnation the contents remain too long in the stomach. This is true both of the test-breakfast and the test-dinner, and of other meals containing much solid food. But a glass of water is evacuated as readily as in health, and this is a differential sign between obstructive and myasthenic stagnation. After a test-meal less fluid is withdrawn than in myasthenia. The stagnation may be of the first degree, the stomach emptying itself after each meal, but later than in health; or of the second degree, the stomach being empty only in the morning before breakfast. During the stage of retention, the stomach is never empty at any moment during the twenty-four hours. Even if the stomach be washed out in the evening, before the Boas supper is given, on the following morning it will be found to contain a noteworthy quantity of food, and this is the pathognomonic sign of retention. In obstructive stagnation and retention the evolution of the diges-

tion of the test-breakfast is abnormal. Not only is digestion prolonged,—as occurs in obstruction, in myasthenia, in hyperchylia gastrica, and in hypersthenic gastritis,—but the lines which represent the evolution of the total acidity of the free and of the combined HCl show sudden rises and falls, which are due to the irregularity in the evacuation of the contents of the stomach. Clinically, two conditions present themselves in obstructive retention, accordingly as the muscular layer is strong or weak. In the one, if the stomach be washed out, two glasses of water will be evacuated within one and one-half hours, as revealed by the cessation of splashing or by the failure to express anything through the tube or to detect anything in the stomach by employing a one per cent. solution of sugar. If the stomach fails to stand this test, myasthenia is not necessarily present, but either the obstruction is great or the muscle is weak. If there is retention in the morning, after the Boas supper has been given on the preceding evening on a clean and empty stomach, and if a pint of water is evacuated within one and one-half hours, there is obstruction, and to that the retention is solely due. In the other condition there is also muscular weakness, and the water is retained much longer when given in the same manner.

A characteristic of the contents of the stomach in retention is the separation on standing into three layers, on account of the gas-forming fermentation—the upper layer being cloudy and frothy; the middle, clear and fluid; and the lower, sedimentary.* The contents withdrawn after the employment of an ordinary diet are watery, highly acid, and fermented; on the addition of grape-sugar they yield large quantities of gas in the fermentation tubes.

The bacteriological signs are variable, but are in keeping with the degree of stagnation and retention and with the quality of the diet. In stagnation, with excessive or active hydrochloric acid secretion, there is chiefly yeast fermentation, and usually formation of acetic acid. If there be no free hydrochloric acid after a test-breakfast, the most common form of fermentation is lactic. In retention the same relation obtains, but when there is excessive and active secretion, hydrosulphuric acid and acetone are sometimes found, and sarcinæ are numerous. If no free hydrochloric acid is present, the organic acids are chiefly lactic and butyric, sarcinæ are rare, and the fermentation is chiefly bacillary. In all forms of retention the germs actively generate gas when the sweetened, unfiltered stomach-contents are placed in fer-

mentation tubes and kept for twenty-four hours at the proper temperature.

Differential Diagnosis and Diagnosis.—During the stage of compensation pyloric obstruction is very likely to be overlooked. Digestion is unconscious, except during the recurring periods of disturbed compensation, when both patient and physician are often satisfied with the common explanation that these attacks are due to indigestion. But whenever a patient has had a disease of the stomach which is liable to be followed by pyloric obstruction, the recurring attacks of motor insufficiency should excite suspicion and lead to a careful examination, and the suspicion should be increased whenever a secretory disorder is absent and there is no trouble in the intestines. These attacks are nearly always produced by dietetic excesses of a particular kind. A heavy meal containing irritants and easily fermentable articles of food may disturb a normal stomach, but more readily one that is myasthenic, or that is only just equal to the task of overcoming a resistance to its evacuation. But recurring attacks of gastric indigestion, accompanied by stagnation and peristaltic pain or vomiting, with little or no fermentation, and not followed by diarrhea or intestinal trouble, and caused likewise by eating a large meal of coarse, solid food, in the absence of fever or of any constitutional disease, are most often due to obstruction. And the probability is greatly increased if the patient has just recovered from an ulcer or from toxic gastritis and if the stomach feels strong to the palpating finger during its contractions, and does not splash longer than it normally should after the administration of a glass of water while it is empty. Dietetic excesses invariably disturb compensation, for the anatomical obstruction is persistent. The detection of a pyloric tumor at once removes all doubt. The possibility of duodenal obstruction producing the same symptoms under the same circumstances should not be forgotten.

Gastric stagnation and retention are either obstructive, myasthenic, or cancerous, and may be due to cancerous or inflammatory infiltration of the muscular layer. Excessive secretion prolongs digestion. The motor power of the stomach in uncomplicated chronic asthenic gastritis is as good as in health. Chronic hypersthenic gastritis may sometimes cause obstruction of the pylorus, and is frequently complicated, particularly in its advanced stages, by motor insufficiency. Cancer may likewise produce the two conditions in both ways. When either stagnation or retention is

chronically present, the search for the causative disease should be made methodically.

1. Is the motor insufficiency due to obstruction or to myasthenia?

2. Is the stagnation or retention due to obstruction or to supersecretion?

3. Is the obstruction pyloric or duodenal?

4. Is the pyloric obstruction benign or malignant?

5. What is the character of the benign process?

6. What is the degree of obstruction?

1. The first step in the solution of the problem is the search for a pyloric tumor. The examination is greatly facilitated by the reduction of abdominal tension through evacuation of the bowels and bladder. The search should be begun when the stomach is empty. With all the viscera thus empty, the examination of the abdomen is made easier. Any abnormality of the abdominal organs is noted, and special attention should be given to the displacement of the right kidney, the distention of the gall-bladder, and the search for any tumor which might obstruct the duodenum. The pylorus should next be sought, using the act of respiration, and also the posture on the left side, in order, if possible, to bring the pylorus within reach. The stomach should next be moderately inflated with air or an effervescent powder, and the pyloric end of the stomach and the position of the whole organ should be noted, as the obstruction may be due to displacement of the stomach. If a pyloric tumor is found, the discovery is very important. Stagnation or retention, with the presence of a palpable pyloric tumor, demonstrates the obstructive nature of the trouble. The presence of the other signs of obstruction, in contradistinction to myasthenia, confirm the diagnosis.

If no pyloric tumor can be felt, a search for a displaced organ or for a tumor which might obstruct the duodenum should be instituted. In the absence of an ulcer history, of toxic gastritis, of the signs of cancer, and of congenital stenosis, and if the pylorus can be felt and presents no tumor, duodenal obstruction is the probable cause.

In the absence of a discoverable pyloric tumor, and of any cause of duodenal obstruction, the differentiation between obstructive and myasthenic stagnation or retention must be made by other symptoms which often have a distinctive and conclusive significance. But at particular moments in the evolution of a number of cases there is a chance to make only a more or less rational guess.

Myasthenic stagnation is a frequent disease of the stomach. Myasthenic retention is rare, and much less common than obstructive retention. In the one or the other degree of motor insufficiency the probability is in favor of the more frequent cause.

If the stagnation or retention has been preceded by a disease likely to produce obstruction, such is probably the genesis and nature of the trouble. In the absence of such a disease myasthenia is probably the cause, and the probability is greater if the patient has gout, or has recently recovered from an acute infectious disease, like influenza or typhoid fever, and if the common signs of myasthenia are present.

The evolution of myasthenia is slow and mild. The trouble may exist for months without producing any local symptoms, and may last for years without the development of supersecretion. Obstruction is usually rapid, painful, and stormy in its evolution. During the stagnation period the stomach often becomes intolerant; and, as a rule, the urine is much less diminished in quantity than it is in myasthenia, and fermentation is much less active. But in the retention period fermentation is active in both diseases, and the difference in the quantity of urine increases.

In obstruction the muscular layer of the stomach often undergoes hypertrophy, and the peristalsis during digestion is palpable, sometimes visible and painful. In myasthenia these signs of increased effort and power are not found.

In myasthenia vomiting is very rare, and pain is nearly always absent during the period of stagnation. In obstructive stagnation both pain and vomiting are very common. During the retention period of myasthenia vomiting is infrequent and copious, but during the same stage of obstruction vomiting, as a rule, is more frequent, occurs oftener as the climax of the trouble excited by a meal, and is accompanied sometimes by nausea and retching, the stomach frequently retracting after its evacuation. Obstructive retention is much more serious and starves more rapidly than does myasthenic retention. Expression of the contents of the stomach is easy in obstruction; it is always difficult and incomplete in myasthenia.

In myasthenia liquids as well as solid food stagnate or are retained. This peculiarity is so pronounced that the disease was once called the "dyspepsia of liquids." In obstruction, on the other hand, liquids are evacuated from the stomach much more readily than semi-solid food. This fact constitutes the differential value of the water-test. In

obstructive stagnation if a pint of water be given when the stomach is empty, it is evacuated within one and one-half hours, which is long before the myasthenic stomach ceases to splash or to yield water upon the introduction of the tube.

Myasthenia often yields readily to treatment, or, in the stage of retention, can often be so managed as to maintain the balance of nutrition at a low level. Obstruction is much more obstinate, and after compensation is broken it is susceptible of little relief except by operation; it is never cured by medication, but its ultimate progress may be thus arrested. Other differential signs are enumerated under the retention form of Myasthenia.

2. The prolonged digestion of hyperchylia gastrica and of hypersthenic gastritis may be confounded with that of obstructive stagnation. In one instance the stagnation is due to excessive secretion; in the other the stagnation is due to obstruction. In both a pint of water is evacuated within the normal time. But the contents of the stomach after the test-breakfast are greater and more fluid, and have less of the roll in supersecretion than in obstructive stagnation. The evolution of digestion is abnormal but regular in glandular gastritis and in hyperchylia gastrica; it is subject to sudden and irregular rises and falls in obstruction, as displayed by the irregularity of the lines which represent the evolution of the total acidity and of the free and combined HCl. In obstruction secretion ceases when the stomach no longer contains food; in the continuous or prolonged secretion the stomach secretes after it has been thoroughly washed out. In obstruction the early morning residual contents may be more acid (H + C) than the contents at the acme of the digestion of the test-breakfast; but this is never so in hypersthenic gastritis not complicated by obstruction or by myasthenia nor in hyperchylia gastrica. The causation and the evolution of these diseases may be widely different, and the results of treatment may be such as are obtained in only the one or the other disease.

3. These many signs and symptoms distinguish obstructive from myasthenic stagnation or retention, and from functional or organic supersecretion. Is the obstruction pyloric or duodenal?

The causes of duodenal obstruction are displacement of the stomach, malignant neoplasms developing in its wall or in the surrounding parts, compression by benign tumors, cicatricial contraction after ulcer or ulceration, local plastic peritonitis, impaction of a gall-stone, and, possibly, a displacement

of the right kidney. The obstruction of the duodenum above the orifices of the pancreatohepatic duct can not be distinguished from pyloric obstruction. But it is in the first part of the duodenum that the traction of a displaced stomach contracts the lumen of the digestive tube; and when the stomach thus displaced is the receptacle of stagnation and retention, and its walls are hypertrophied, and no tumor of the pylorus can be felt, and when there are no signs or history of ulcer or cancer of the stomach, it is then reasonable to conclude that the obstruction to the evacuation of the stomach is in the duodenum, and is due to the traction and bending. Obstruction below the opening of the common duct produces special signs. The clinical history may locate the trouble at this point, which may be more sensitive to pressure than are the other parts of the abdomen. The history of gall-stones or of duodenal ulcer, and the existence of a circumscribed tender point in the back and to the right of the lower dorsal vertebra, are of particular value in this connection. Search should be made, also, for a distended gall-bladder and for cancer of this organ. The vomit and the contents of the stomach furnish signs of unquestionable value. The vomit almost continuously contains bile, and, indeed, the bile is present in the first mouthful brought up, showing that it is not pressed into the stomach by the act of vomiting. No significance should be attached to the presence of bile in the last of the matter vomited; but this sign is very valuable, when repeatedly obtained, in excluding obstruction of the pylorus or of the first part of the duodenum. In duodenal obstruction of a high degree the duodenum itself is the site of retention. If the stomach be thoroughly washed out, the later vomiting of the duodenal contents or their removal through the tube after massaging the duodenum is a very valuable sign. It should not be forgotten that it is very difficult to cleanse the stomach completely when food is retained; but this procedure is much easier when, as is most frequent in obstruction, the stomach is not myasthenic. Another sign is the great variability of the free hydrochloric acidity of the gastric contents, which is due to the variable quantity of the duodenal contents regurgitated into the stomach. The vomit also contains pancreatic juice, except when the pancreas is diseased. The presence of pancreatic juice in demonstrable quantity excludes advanced disease of the pancreas, and consequently it also excludes one of the causes of duodenal stenosis. The frequent vomiting of duodenal contents in association with gastric retention is in favor of obstruction in

the lower part of the duodenum, and aids in the exclusion of obstruction of the pylorus. Duodenal obstruction being excluded, the benign or malignant nature of the pyloric obstruction should next be determined.

4. The differentiation of benign and malignant obstruction of the pylorus may be easy or exceedingly difficult, and is sometimes impossible. If the obstruction develops as a sequel of ulcer or of toxic gastritis, the benign and cicatricial nature of the obstruction is almost certain, for, practically, the cancerous degeneration of ulcer is so rare that it may be disregarded. Where a knotty, hard tumor can be felt there is most probably carcinoma; and there is little room for doubt if the functional, bacteriological, blood, and nutritive signs of cancer are present. But too often the clinical history and the signs are not so distinctive.

The differentiation is frequently dependent on a predominance of probabilities, so that only a rational guess at the truth can be made. If hydrochloric secretion is normal or excessive, the disease is probably benign, but it should not be forgotten that carcinoma of the pancreas and gall-bladder seldom diminishes gastric secretion before the period of cachexia arrives. Lactic acid formation is not a pathognomonic sign, as it may occur in benign retention accompanied by asthenic gastritis. But the weight of evidence is in favor of cancer where there is no free hydrochloric acid and where the lactic acid is formed by bacilli. The formation of hydrogen sulphid is also a benign sign. The continued presence of sarcinæ for a long period does not occur in carcinoma of the pylorus, this germ disappearing rapidly when lactic acid begins to develop. Butyric acid fermentation is also more frequent in carcinoma. The functional and bacteriological signs do not rapidly change in benign obstruction, as they do during the rapid evolution of carcinoma. These signs may be so grouped as to produce a preponderant weight of evidence in favor either of the benign or the malignant nature of the obstruction. The functional and bacteriological signs are of less value when it is not certain that the obstruction is pyloric and that it is primarily so.

The age of the patient is of very little value, except in the exclusion of congenital atresia or stenosis, if the trouble began after childhood. The age being below twenty is against cancer. The evolution of cancer is rapid and progressive, presenting only short periods of improvement. There is no long period of gastric trouble followed by a period of compensation before the development of stagnation

and retention. Rectal feeding is less beneficial than in benign obstruction, and careful feeding by mouth and rectum is powerless against the progressive loss of strength, the excessive albuminous waste, and the toxic leukocytosis of cancer. The cachexia of benign obstruction is dry, like that of simple starvation, and with it is never found the fugacious edema of malignant disease.

The dissemination signs of cancer should be carefully sought, and enlargement of the abdominal and supraclavicular glands and secondary nodules in the liver should be considered conclusive.

The water-test is also of some differential value in the stage of stagnation, water being evacuated much more rapidly in benign than in malignant disease.

In cases where only a few of these distinctive features are found, the diagnosis should be held in suspense. Cases beginning suddenly, developing rapidly, with pain and circumscribed tender points, with stagnation or retention, with the presence of free acid, yeast, and sarcinæ, without a palpable tumor or with one that is not characteristic of either cancer or ulcer, may be due either to ulcer or to cancer. Under these circumstances the most rational but often incorrect guess is obstruction due to ulcer.

A positive diagnosis should be given only when the weight of evidence is overwhelming; under other circumstances a probable opinion or no opinion whatever should be expressed, if it be desired to avoid the unpleasant revelations of an exploratory laparotomy. An accidentally correct guess possesses no merit, and a false opinion would only yield chagrin and condemn immodesty.

5. The causes of benign obstruction are ulcer, ulceration, benign tumors, foreign bodies, arrested development, hypertrophy, and hyperplasia. The cicatricial obstruction of toxic gastritis is revealed or excluded by the clinical history. Syphilitic and tubercular ulceration and obstruction by benign tumors and foreign bodies are not recognizable. Congenital atresia and stenosis are revealed by the age when they begin, by their evolution, and by the signs and symptoms, already described, which they produce. The hypertrophy and hyperplasia are characterized by a preceding productive gastritis in the inflammatory form, and by the presence of a smooth, regular, contracting and relaxing, and non-adherent tumor, through which the gastric contents spurt and bubble. The evolution may be rapid after the second degree of stagnation develops. The most common form of benign obstruction is

caused by ulcer, and its only distinctive signs are those of the ulcer which precedes or accompanies it.

6. The diagnosis of the degree of obstruction is easy. During the period of compensation the stomach empties itself in about the normal time, without the development of an excessive germ growth.

The period of stagnation is characterized by the delayed evacuation of the stomach. The test-breakfast and the Leube-Riegel dinner remain longer than the normal period in the stomach, as revealed by the employment of the tube and by splashing sounds, particularly during the relaxation of the walls of the organ. Two degrees of stagnation may be distinguished, and are clinically important. In the mild form the stomach empties itself after each of the three meals. In the severe form the stomach is found empty and free from splashing only before breakfast.

In retention the stomach never completely empties itself, and splashes before anything is taken into it in the morning. Clinically, a particular test should be adopted as the criterion. If the stomach be thoroughly washed out in the evening and the Boas supper given, food should be found in it the following morning.

The degree of retention is revealed by the quantity of urine passed in the twenty-four hours, provided there is no great loss by perspiration or by an intercurrent diarrhea, and provided, further, that the kidneys are not diseased, the stomach not intolerant, and that three pints of fluid be taken during the day. Another rough measure is the quantity and composition of the feces. The progress of starvation is still another guide.

Prognosis.—Pyloric obstruction is a very serious disease, and the prognosis is bad. The greater the obstruction, the worse is the outlook. Naturally, the nature and rapidity of development of the causative trouble are modifying factors. Medical treatment is palliative; surgical treatment may be curative.

Treatment.—The treatment of pyloric obstruction is medical and surgical.

The medical treatment is only palliative, but is none the less valuable before an operation is indicated, and also when surgical intervention is not advisable on account of the possibility of maintaining nutrition by the administration of the proper foods in the right manner.

The diet is the most important part of the treatment. During the stage of compensation digestive hygiene and the

avoidance of dietetic excesses and errors are imperative as prophylactic measures. The diet should consist of the best meats—like beef, mutton, chicken, white meat of turkey, pheasant, grouse, quail, squab, and lean fresh fish. The cereals, particularly the preparations of wheat and rice, thoroughly cooked, are also suitable. Vegetables should be prepared as purées. No fat, except butter or cream or Hauswaldt's "vigor chocolate," should be permitted, and the sweets should be of the simplest sorts. Stewed fruits are permissible in moderation, and, likewise, the preparations of eggs and milk, on condition that they are well borne. The diet in this stage is prophylactic, and in addition to being nutritious, digestible, and utilizable, should make the muscular work of the stomach as light as possible, and not irritate the mucous membrane mechanically or chemically nor favor germ growth. Fine mechanical subdivision, either by previous preparation or by thorough mastication, is an absolute and invariable requisite. A dry diet is a mistake in any stage of obstruction; and during compensation coffee, weak tea, cocoa, and, if it has been the habit of the patient to take alcoholic drinks, a little old whisky diluted, or old and light wine, should be allowed in such quantity as not to exceed the limits of good hygiene. There is no tendency to water-stagnation or retention as in myasthenia, but coarse, irritating, and solid food is liable to disturb compensation. No incompletely fermented nor yeast-containing drinks of any sort should be permitted. During the acute breaks in compensation the stomach should be washed out and given functional rest for twenty-four hours, and the feeding by mouth should be resumed with small quantities of liquid food. There is, during this stage, no indication for drugs, and the stomach should be particularly guarded against medication liable to derange its secretion. The general health should be maintained by hydrotherapy and by good general and digestive hygiene.

Stagnation is a sign of danger, and demands strict care and efforts to restore compensation or to check the advance of motor insufficiency. The diet for the stage of compensation should be modified by the exclusion of all food which easily ferments, and sweets, consequently, should be entirely forbidden in many cases. The food for the twenty-four hours should be divided into three equal portions, and so limited in bulk as to obtain the complete evacuation of the stomach before the next meal. The nourishment should be fluid, and the

liquids should be so increased in proportion to the solids as to obtain this end. Rectal feeding should be employed exclusively, during the attacks of intolerance. Strychnin, massage, electricity, and general neuromuscular tonics should be used to restore compensation or to control the increasing insufficiency of the muscular layer. In the mild form of stagnation there is no indication for stomach washing, unless there be excessive fermentation. But in the severe form of stagnation the stomach should be washed out daily with a weak alkaline solution if there be much yeast or sarcinæ. The preferable time for the stomach washing is in the early morning before breakfast, so as to remove no nourishment. But if the fermentation can not be thus controlled, the lavage should be performed in the evening with Thiersch's solution, and then with boiled water to remove the residuum of the solution. Three grains of resorcin resub. dissolved in a tablespoonful of chloroform-water should be left in the stomach over night.

During the period of retention the diet of stagnation should be continued, and the stomach should be daily washed out in the morning before breakfast with water alone, or with an alkaline solution, or with Thiersch's solution and water. A solution of hydrochloric (1 : 1000) acid should be left in the stomach if too little of it is secreted; or, in case it is secreted in excess, the stomach should be left empty, or a solution of nitrate of silver or of resorcin, or of any non-irritant anti-fermentative, should be left in the organ. Rectal feeding should be methodically employed.

Three grand principles control the feeding of a patient with obstruction of the pylorus.

1. The stomach must be empty at the beginning of each meal.

2. All the food must be very finely divided, readily soluble or easily rendered fluid, and without unsuitable action on secretion or on the motor function.

3. The diet must be varied and sufficient to support or to improve nutrition.

If the first principle is violated, and the stomach is not empty at the beginning of each meal, the symptoms will not be relieved and there will be no improvement. In the mild form of stagnation there is no chance to violate this rule. In the severe form of stagnation it may be only necessary to separate the meals by intervals which are longer than our ordinary dietetic habits make them, or it may be necessary

to restrict the number of meals to two in the twenty-four hours. As the stomach regains its tone and power a light lunch may be permitted between breakfast and dinner, but it must consist of food which rapidly leaves the normal stomach and which can be easily evacuated through the narrow pylorus. In obstructive retention the two-meal system should be adopted without delay, and if the stomach is not empty an hour before these two meals, the stomach-tube must be used to render it so. The contents should be removed, the stomach washed out and left empty.

The physical properties of the food are of the utmost importance. The pyloric opening is small, and all the food must be such as can easily pass through a small canal. Naturally, the state of secretion should control the choice of the articles of the diet, and it may be necessary to embody other dietetic principles in the management of particular cases. But the minute division and the solubility or the fluidity of the food after its preparation are essential properties, which may be readily secured by the method of cooking or by straining the food after it is cooked. The crushed and cooked muscle pulp of meat, eggs but slightly cooked or hard boiled and powdered, fresh fish with loose and short fibers, milk if it agrees well, cream, butter, cheese, zwieback, light cake, cereal (strained) puddings, light custard, oatmeal (strained), cornmeal mush, corn bread made of "round" cornmeal, rice passed through a colander, rice cakes, flour "ball," vegetables passed through a sieve, cooked fruit strained, and similar articles from all the grand classes of food may be ordered. Fed in this manner, patients with pyloric obstruction often improve rapidly and are able to eat and digest a varied diet comfortably.

The symptoms requiring special medication are vomiting, pain, and constipation. If the pain and vomiting are not relieved by the diet and lavage, and by functional rest of the stomach obtained by exclusive rectal feeding, codein and atropin should be given hypodermically and a hot or Winternitz compress should be placed over the abdomen. Counter-irritation and the locally-acting drugs are useless against vomiting and pain. Bismuth, in a large daily dose when the stomach is empty, is an exception to this general rule. A sedative to both nerve and muscle should be used, and the codein and atropin form the most beneficial combination. The constipation should be treated exclusively by clysters of water, of soapsuds, of water and glycerin, or of water,

glycerin, and oil. The patient should remain quiet in bed, so as to reduce the nutritive needs of the organism and to conserve the ever-failing vital energy.

The only hope of a possible cure of pyloric obstruction lies in surgery. Numerous operations have been advocated and performed. Loreta's digital divulsion is not a good operation, since it is often inefficient, only practicable in a few cases, and about as dangerous as the operations which give greater hope of benefit. Gastrostomy and a permanent pyloric tube, through which the patient is fed, can not be said to decrease very much the discomfort of the patient. Bernay's curetting operation has justly found but little favor. Duodenostomy has proved objectionable. Jejunostomy has only one indication—cancerous degeneration of the body of the stomach, rendering gastro-enterostomy impracticable. There remain pylorectomy, pyloroplasty, gastro-enterostomy, and combined pylorectomy and gastro-enterostomy.

The choice of an operation is determined by the nature of the obstruction, the existence of adhesions, the degree of atrophy of the muscular layer, and the strength of the patient. For localized and unadherent carcinoma, pylorectomy and combined pylorectomy and gastro-enterostomy are the operations to be chosen. If the carcinoma is already disseminated or adherent, gastro-enterostomy is the proper palliative operation. The ideal operation for cicatricial stenosis is pyloroplasty. In case it is not practicable on account of adhesions or induration and thickening, gastro-enterostomy should be performed.

Indications for an operation are present much earlier than are usually admitted by the physician or accepted by the patient, who, as a rule, gives his consent only when death stares him in the face. An operation should be advised in cancerous obstruction as soon as the nature of the obstruction is recognized, and the earlier it is done, the better are the chances of prolonging life and making the patient more comfortable. Continuous obstructive retention, however caused, demands surgical intervention as soon as the stomach can be prepared for operation, especially if a judicious effort has failed to restore compensation to such a degree as to render it possible to nourish the patient. The same rule holds good in the severe form of obstructive stagnation accompanied by much suffering, or by the first stages of starvation, or by rapid progress of the disease. Operation should be deferred in the mild form of benign obstructive stagnation.

The reëstablishment of the permeability of the digestive tube relieves all the symptoms due to the stagnation and retention—the appetite improves, the patient gains weight and strength, the vomiting and pain cease. But in carcinoma the secretory function is not restored, a result due in all probability to glandular degeneration or transformation. The motor function is restored and the enlarged stomach retracts, unless the disease is so far advanced as to produce atrophy or degeneration and infiltration of the muscular coat.

SECTION VI.

THE VICIOUS CIRCLES OF THE STOMACH.

CHAPTER I.

OTHER ORGANS IN THE DISEASES OF THE STOMACH, OR THE STOMACH IN THE CAUSATION OF DISEASE.

THERE is no doubt that the diseases of the stomach sometimes play an important part in the causation of disease, but we consider it true, also, that the effects of a diseased stomach are often overestimated. Probably no other organ more frequently becomes diseased secondarily, but the vicious circles established between the stomach and the other important organs of the body seldom, in comparison, begin in the stomach. We strongly oppose the theory which makes the diseased stomach an all-important disease factory.

Many of the effects of diseases of the stomach are merely passing and variable symptoms; but other effects are more persistent and constant. It is these constant and persistent effects which we wish to describe, constituting complications or sequelæ linked in close causal relation with the disease of the stomach.

The stomach as a disease-producing organ may act in several ways—through its influence on intestinal digestion and on nutrition, through its disturbance of physiological chemistry, and through auto-intoxication. The influence on intestinal digestion is direct, the abnormal chyme producing abnormal chylification. On all other organs the diseased stomach acts either through the nervous system or through the blood. It is difficult to determine how much disturbance is due to reflex action; it is difficult to know how far the peripheral irritation involves the sympathetic and cerebrospinal centers; it is difficult to discover how far the chemistry of the body is altered in a particular case by the diseased stomach; and it is far more difficult to estimate with precision the injury done by auto-intoxication.

The existence of gastric auto-intoxication is only an infer-

ence, and the correctness of the inference is denied by some writers. It is claimed that the total quantity of the fermentation acids formed in the stomach is not great; that few of them are toxic, even in large quantity; that they are combined and diluted by the blood, that some of them are so harmless as to serve as food; and that putrefaction, which is the chief source of poisons, occurs in the stomach seldom and irregularly and, indeed, almost accidentally. Clinical and experimental evidence is not conclusive, it is true, as to the existence of gastric auto-intoxication, but it would seem that iconoclasm threatens to go too far. The toxicity of the gastric juice is much greater in some of the diseases of the stomach than in health; the toxicity of the urine is often increased in the diseases of the stomach accompanied by toxemic symptoms, although the bowels be healthy and the liver show no signs of functional insufficiency; there are often present in the diseased stomach, and, indeed, in a state of active and virulent growth, germs which produce poisons in cultures. These are conclusions deduced from our investigations. Hydrogen sulphid formed in the stomach and found in the breath and in the urine gives rise to a special symptom-group on its way through the system. Butyric acid certainly produces local irritation and systemic symptoms. Acetone is sometimes found in the stomach when oxybutyric acid and acetone are found in the urine. Moreover, the toxicity of the urine is no index of the toxemia, for gastric poisons are not eliminated by the kidneys only, and it is probable that some of them, at least, are changed into simpler and non-poisonous compounds during their passage through the body. Furthermore, the absence of a perceptible increase of the toxicity of either the gastric contents or the urine does not exclude the existence of slow, chronic self-poisoning. The denial of the existence of gastric self-poisoning is based on a simple negation, and can not be justly made in disregard of the clinical and experimental evidence which we have, however little it may be.

I. INFLUENCE ON THE INTESTINES.

The functions of the intestines may be disordered indirectly by the injurious influence of the diseases of the stomach on the nervous system, on the liver, on the blood, on nutrition, on the circulation, and on the kidneys; but there can be no question that their most direct action is on the intestines by reflexes originating in the stomach and conveyed by the

sympathetic and the pneumogastric nerves. These influences, however, are difficult to define, and they may be dismissed with this brief notice in order that we may pass at once to the consideration of the more important disturbances which occur in virtue of the close association of the stomach and intestines in digestion, and of their being but divisions of the digestive tube with one grand work in common.

Whenever the stomach fails to do its required digestive work an additional burden is thrown on the intestines. If the nitrogenous food is not properly peptonized, and if the bundles of muscular fibers are not unbound, as is the case in insufficiency of the secretory and motor functions, the omitted work must be performed in the bowels. If the action of the saliva is too rapidly arrested in the stomach, the intestines must convert more than their share of the carbohydrates. The healthy intestines, as is well known, are capable of performing this extra work perfectly, and the digestion and the utilization of the food may be as good as when both the stomach and intestines do their work normally. But the extra work is a menace to the integrity of the functions of the intestines, and their susceptibility to disease is increased.

A disease which disorders the digestive work of the stomach, disorders, as a necessary consequence, the secretion and peristalsis of the intestines. It is well known that the saliva and the products of salivary digestion are physiological excitants of gastric secretion. If the saliva be excluded from the food and its entrance into the stomach prevented, the activity of the secretion of hydrochloric acid and the two gastric ferments during the digestion of the test-breakfast is reduced to about one-half of what it should be. The products of gastric digestion, also, excite the functions of the stomach. Chyme, on the other hand, as it undergoes conversion into chyle, is the physiological excitant of intestinal secretion and peristalsis, so that an alteration of the composition of the chyme must entail an alteration of intestinal secretion and peristalsis. This alteration may be compensatory or it may be so great as to produce functional insufficiency.

Now, functional insufficiency of the intestines, whether it be produced by extra work or by disordered gastric secretion and peristalsis, favors intestinal fermentation and putrefaction, and these in turn become active enemies of intestinal health. Thus the diseases of the stomach which are accompanied by disorders of secretion (excessive or diminished) and by disorders of the motor function (excessive or diminished) produce naturally disorders and diseases of the intestines.

If the disease of the stomach be accompanied by excessive gastric secretion, the carbohydrates must be digested by the bowels, and the gastric digestion of albumin is very active. If sweets are not excluded from the diet, gastric fermentation may become active, and, continuing in the intestines, it may prevent intestinal putrefaction; but even in this condition putrefaction is likely to begin in the cecum, where the contents first become nearly neutral or alkaline. The colonic putrefaction is not accompanied by indicanuria, but it is manifested by very foul stools, and by the excessive formation of H_2S gas. If sweets be excluded from the diet, gastric fermentation may be controlled, but intestinal putrefaction then begins high up in the small bowel, and the contents become thoroughly rotten in the colon. Indicanuria is very marked, and putrefaction and pancreatic superdigestion are only favored by the excessive gastric peptonization in the same manner as when predigested foods are eaten. If the disease of the stomach be accompanied by diminished gastric secretion, salivary digestion is very active and the digestion of the albumins is thrown on the bowels. Fermentation begins high up in the small intestines (even in the stomach, if there be motor insufficiency), and continues as long as the intestinal contents contain fermentable matter. Putrefaction does not occur in the small bowel, and the fermentation is usually active enough to prevent excessive putrefaction in the colon. If gastric stagnation or retention be present, there will be intestinal putrefaction or fermentation according to the reaction of the chyle and its richness in peptones or carbohydrates. Consequently, in diseases of the stomach which are accompanied by disorders of secretion and by disorders of the motor function, intestinal secretion and peristalsis may be disturbed, the food may be lost by fermentation and putrefaction (the analysis of the stools showing only an apparently normal utilization), auto-intoxication may be produced, and enteritis, colitis, or enterocolitis may result. Diminished gastric secretion also favors enteric infection.

Finally, gastroptosis may be primary and cause enteroptosis, and this process is but little less frequent than the production of gastroptosis by the primary prolapse of the colon. Gastro-enteroptosis may induce chronic changes in the abdominal sympathetic and in the nutrition of the intestines. Enteritis membranacea may then be an ultimate effect.

II. INFLUENCE ON THE LIVER.

The manifold functions of the liver—the largest and most important gland of the body—may be disordered by diseases of the stomach.

The liver is an organ of assimilation and disassimilation. This nutritive function may be disordered by the products of abnormal digestion and by auto-intoxication, or it may be disturbed on account of the irritation, or the overwork, or the required performance of unusual work.

The liver is also a poison-destroying organ. It accumulates, eliminates, and destroys the poisons which are carried to it by the portal vein. This function can be disturbed by auto-intoxication and by the absorption (without conversion) of the products of peptonization. Peptones and digestive albumoses, when they get into the circulation without being first transformed, act as poisons.

The liver is also a digestive organ, for the bile exerts a marked influence on the intestinal digestion and utilization of food. The quantity and activity of its secretion is dependent in part on the quantity and quality of the food and on the composition of the chyme, a large mixed meal when it is normally digested by the stomach being the most active cholagogue known. This physiological and purposive secretion of bile may be disordered by the diseases of the stomach which require a modification of the diet as regards the proportion of the grand classes of food which enter into its composition, which withhold, by retention or by vomiting, the required quantity of food, and which alter by secretion, digestive transformation, fermentation, and putrefaction the normal properties of the chyme.

The liver is a blood-destroying organ, and it has probably something to do with blood building and with the maintenance of the composition of the plasma. This function may be disordered indirectly by the diseases of the stomach through their action on the blood (hematocytolysis) and through the association of disordered functions. If one function of the liver becomes disordered, the other functions may be compromised with it.

Consequently, the functions of the liver may be disordered by disease of the stomach. Hepatic insufficiency may be thus established, or the organ may become congested and, finally, inflamed. The congestion and inflammation may be acute or chronic. It is likely that the functions of the liver

may be deranged by reflexes from the diseased stomach, in the same manner that gall-stones produce hyperchlorhydria and hyperchylia gastrica, or that they may be deranged by the action of the diseased stomach on the cardiovascular system. Carcinoma and ulcer of the stomach may produce hepatic complications.

III. INFLUENCE ON THE BLOOD.

The injurious influence of diseases of the stomach on the blood is exerted in several different ways:

1. Diseases of the stomach frequently produce inanition-anemia, which is the sequel of subnutrition. Inanition acts in two principal ways on the blood. It may produce insufficiency of the hematopoietic organs, just as it produces general functional inactivity; or it may alter the composition of the plasma so that it becomes poor in nitrogenous matter, the resistance of the red corpuscles being diminished and the development of the white corpuscles being decreased. Consequently, subnutrition may produce dyshematopoietic oligocythemia, or the dyshematopoiesis may be accompanied by hematocytolysis, as is the case in the grave anemia of atrophy of the gastric glands.

2. The diminished supply and the diminished absorption of water, and its increased elimination by supersecretion or vomiting, may produce oligemia sicca. The oligemia may be simple, the red corpuscles being normal in number, in coloring (hemoglobin), in size, in form, in resistance, and in their affinities for stains; or the oligemia sicca may mask a disease of the red corpuscles, the deception being quickly made plain by the use of the microscope.

3. The loss of blood by hemorrhage (ulcer, carcinoma, erosions) produces an acute or chronic anemia, which may be mild, severe, or grave. Oft-repeated small hemorrhages produce a grave and rebellious form of anemia when the daily or frequent losses of blood exceed the quantity of blood supplied by the hematopoietic organs.

4. Auto-intoxication does more direct and more extensive injury to the blood than it does to the nervous system, the cytoplasmic poisons destroying the red corpuscles and the activity of the blood-building organs are decreased. Consequently, the anemias of auto-intoxication may be dyshematopoietic or hematocytolytic.

5. The blood may also be injuriously affected by the influ-

ence of gastric secretion or retention on the percentage of salines in the blood. The sodium chlorid is most reduced in this manner, and the plasma may thus become hematocytolytic.

6. Clinical observation teaches that chronic irritation of the abdominal sympathetic exerts a depressing influence on the regeneration of the blood, particularly as regards the development of the hemoglobin of the red corpuscles. The action of the irritable abdominal sympathetic (gastroptosis, adenohypersthenia gastrica, neurasthenia gastrica) is the most common cause of simple and of chlorotic oligochromemia.

7. Peptonization and peptone absorption are causes of physiological leukocytosis, and in the advanced stages of some of the diseases of the stomach this digestive leukocytosis is not excited. As a consequence the resisting power (healing and phagocytosis) of the system is diminished, and the normal quantity of albumins in the plasma is not maintained. Leukopenia may be produced by subnutrition or by the absorption of the unconverted products of albumin digestion.

8. Pathological leukocytosis may develop in carcinoma and in the diseases of the stomach with inflammatory complications.

As regards the diseases of the red corpuscles, diseases of the stomach may produce either oligochromemia or oligocythemia. Oligochromemia is always dyshematopoietic, unless it represents the regeneration period of oligocythemia. The disease and the symptom may be readily distinguished by the clinical history and by the microscopic and staining properties of the blood. Oligocythemia, on the other hand, may be dyshematopoietic, hematocytolytic, or degenerative. The diseases of the stomach never cause the red corpuscles to undergo primary degeneration. Consequently, oligocythemia of gastric origin is always due either to insufficient and defective development of the blood or to the excessive destruction (or loss by hemorrhage) of the red corpuscles. These two forms of oligocythemia may be readily distinguished by the signs of dyshematopoiesis and by the signs of hematocytolysis, a discussion of which here would lead too far from the original subject.

Of the diseases of the white corpuscles, leukemia is never produced by the diseases of the stomach. But digestive leukocytosis may not occur, pathological leukocytosis may be persistently present, the white corpuscles may degenerate (as many as ten per cent. of them may display the degenerative changes), or leukopenia may represent the chief alteration of

the blood. The plasma may be altered qualitatively or quantitatively and become as a consequence hematocytolytic, or the total volume of the blood may be decreased.

The changes of the blood which are produced by the diseases of the stomach are not so constant or so characteristic as to enable us to reason back from the disease of the blood, to the particular disease of the stomach which has caused it; but a particular disease of the blood should direct the search for the group of diseases of the stomach which may cause it, and the blood changes have some diagnostic value in the differentiation of one disease or group of diseases of the stomach from another. The blood changes produced by the particular diseases of the stomach have been described under the symptomatology of those diseases.

IV. INFLUENCE ON NUTRITION.

Not every disease of the stomach disturbs nutrition. In some cases the digestion and utilization of the food may be normal; assimilation and disassimilation may go on as in health; enough food may be ingested and retained to supply the needs of nutrition. But such is not always the case, not even in the dynamic affections of the stomach nor in the mild forms of its anatomical diseases. The acute diseases of the stomach may rapidly affect nutrition, and the chronic diseases seldom run their long course without producing subnutrition or without disturbing the processes of nutrition. The most frequent of these disorders of the processes of nutrition are excessive nitrogenous waste, uricemia, and phosphaturia.

Subnutrition may be caused by a disease of the stomach in a number of ways, and it may vary in degree and in the rapidity of its development. The diet is often insufficient on account of loss of appetite, on account of disgust for some one of the grand classes of food, on account of a desire to avoid the pain or discomfort of digestion, or on account of an injurious plan of alimentation. Moreover, when the alimentation is sufficient, a portion of the food may be lost by vomiting, by fermentation, by putrefaction, by diarrhea, or by failure to digest and absorb it to the same degree as in health. In the advanced cases of anorexia nervosa the patient presents a picture of slow and self-inflicted starvation, and the loss of appetite in the anatomical diseases like carcinoma, chronic asthenic gastritis, and acute gastritis

is one of the causes of subnutrition. The pain of ulcer, of chronic hypersthenic gastritis, of adenohypersthenia gastrica, or of carcinoma, the discomfort of neurasthenia gastrica, and of hyperesthesia gastrica, and of the diseases accompanied by retention and by stagnation, often force the patient to diminish the quantity and to contract the variety of the food below the requirements of the body. Moreover, chronic pain itself exerts a depressing influence on digestion and is a cause of emaciation. Frequently repeated alimentary vomiting, whether it be nervous, central, or reflex, or symptomatic of a disease of the stomach, may produce subnutrition as surely as does the failure to eat enough to support the body. In myasthenia gastrica and in pyloric obstruction—briefly, whenever motor insufficiency exists—nutrition is in more or less danger; if there be only stagnation, the influence on nutrition is determined by the degree of fermentation of the chyme; while if there be retention, the body may starve for lack of both food and water. It is well known that the intestines are capable of doing all the digestion and absorption that the body requires, but motor insufficiency may withhold the opportunity to establish digestive compensation. The disturbances of secretion may also produce subnutrition. If secretion be excessive,—as in adenohypersthenia gastrica, in chronic hypersthenic gastritis, and in ulcer,—the digestion of the starches is interfered with in the stomach, and the excessive peptonization of the albumins may increase intestinal putrefaction and produce intestinal irritation and diarrhea. Nature here does what the physician often causes when he prescribes the digestive ferments and peptonized foods. If secretion be diminished, the albumins may not be properly digested, but the salivary digestion of the starches is more active than in health, and the intestines, if they be healthy, are likely to establish digestive compensation. But it often happens that loss of appetite, pain, faulty alimentation, vomiting, disorders of secretion, motor insufficiency, fermentation, and an abnormal chyme act more or less in concert, and produce subnutrition which is more or less rapid and grave.

Excessive nitrogenous waste may be caused by a disease of the stomach. In subnutrition not only the body fat but also the body protoplasm is destroyed. The organism eats itself—lives on itself. But in carcinoma, in some cases of acute mycotic gastritis, and, probably, in some forms of gastric auto-intoxication, nitrogenous catabolism is in excess of the requirements of the body. The hyperazoturia manifests

a purposeless waste of albumin. The inference seems plausible that the excessive destruction of cellular protoplasm is due to protoplasmic poisons formed in the neoplasm or in the contents of the stomach.

The diseased stomach may cause accumulation of **uric acid** in the system by producing acid auto-intoxication. The so-called uric acid diathesis is not a morbid entity with one cause, but it is a chemical condition which is variable in its manifestations and complex in its causation. The accumulation or precipitation of uric acid in the organism is the result of pathological chemistry, and is the expression of a series of diseases which are commonly classified as gouty. There is no insufficient oxidation, for uric acid is the end product of nuclein waste. There may or may not be an excessive formation of uric acid. There may or may not be retention of uric acid as a result of the insufficiency of the eliminating organs. There may be no quantitative anomaly, but only a change in the form in which the uric acid exists, as a result of the altered reaction and composition of the fluids of the body. It is probable that uric acid circulates in the body as a quadriurate, or as a biurate in combination with the neutral phosphate of soda. Be this as it may, the precipitation of the uric acid is prevented by the accompaniment of a sufficient proportion of the neutral disodic phosphate. If the biurate or quadriurate increases proportionately beyond a certain limit, or if the neutral phosphate of soda decreases beyond a certain limit, there will be precipitation. Consequently, the precipitation of the urates is inaugurated either by a diminution of the available neutral phosphate or by an increase of the uric acid. Furthermore, the available neutral phosphate may be decreased by a diminished supply of protecting alkalies or by an increased supply of converting acids. We therefore recognize three grand forms of the uric acid diathesis or uric acid precipitation :

1. Gastro-intestinal form.
 (*a*) Acid fermentation.
 (*b*) Excessive pancreatic and intestinal secretion.
 (*c*) Diminished gastric secretion.
2. Nutrition form (excessive formation of acids : phosphoric and organic, or uric acid).
 (*a*) Defective alimentation.
 (*b*) Excessive catabolism.
3. Retention form (defective elimination of acids).
 (*a*) Insufficiency of the skin.
 (*b*) Insufficiency of the kidneys.

The gastro-intestinal form of the uric acid trouble is due to the excessive acidity of the system and to the resulting diminution of the quantity of the neutral phosphate of soda which is available for holding the uric acid compounds in solution. The stomach removes the chlorin to form hydrochloric acid, and leaves the alkaline base of the chlorid for the protection of the neutral phosphate. The excessive secretion of the intestines and of the accessory digestive glands removes too much of the protecting alkalies. Fermentation produces acid self-poisoning. The result is excessive acidity or diminished alkalinity of the fluids of the body, diminution of the available neutral phosphate of soda, and precipitation of the uric acid compounds. Ninety grains of bicarbonate of soda administered before breakfast is sufficient, in health, to neutralize the acidity of the system and to render the urine secreted during the following two hours neutral in reaction. If the urine, on making this test, does not become neutral, its acidity is the exact index of the excessive acidity of the system. The gastric form of gout results only from a chronic disease of the stomach, and is most frequent when the diminished gastric secretion and gastric fermentation are accompanied by excessive intestinal secretion. Chronic asthenic gastritis with motor insufficiency, carcinoma, and myasthenia with diminished secretion are the most common diseases of the stomach which may produce uric acid precipitation and retention.

As with uric acid in the uric acid diathesis, so is it with phosphoric acid in phosphaturia: it is not the quantity, but the form, of the uric acid and of the phosphates which is important. The acidity of the urine is diminished until it is nearly neutral, or neutral or even alkaline, and the phosphates precipitate in it either spontaneously or on heating. The quantity of the acid phosphates is diminished and the quantity of the neutral and alkaline phosphates is in excess. Instead of increased there is diminished acidity of the fluids of the body, which is not incidental to alimentation, though it may be produced and controlled by it, but which is a chemical condition due to excessive elimination of acids, accompanied often by diminished removal of alkalies by the intestines. During the period of normal gastric digestion the acidity of the urine is diminished, and this condition of the urine results from the withdrawal of acid from the body by gastric secretion. The degree of change of the acidity of the urine during digestion, and under the same conditions, is a rough index of the amount of acid secreted by the stomach.

In the diseases accompanied by subacidity the diminution of the acidity of the urine is less than in health, and in the diseases accompanied by superacidity the diminution is greater than in health. If the superacid gastric secretion be removed by vomiting or by lavage, the diminution is still more marked, and the total urine of the twenty-four hours may be milky (phosphates), alkaline, and poor in chlorids. Gastric phosphaturia is found in chronic hypersthenic gastritis, adenohypersthenia gastrica, and in the other diseases of the stomach accompanied by superacidity or by supersecretion, whether they are or are not associated with stagnation, or retention, or vomiting.

V. INFLUENCE ON THE HEART AND CIRCULATION.

If it be borne in mind how fine and complex is the mechanism of the circulation, how numerous are the influences which control or alter the caliber of the arterioles and the action of the heart, it should cause no wonder that the heart is frequently disturbed by the pathological reflexes and the mechanical compression of an organ so closely situated and so intimately connected with it by an almost common nerve-supply as is the stomach. Normal digestion increases the frequency and strength of the heart-beats, and strong excitation of the mucous membrane of the stomach makes the heart's action slow. The close anatomical and physiological relationship would seem to furnish good grounds for expecting the diseased stomach easily and frequently to disorder the heart's action. This natural expectation is only in part fulfilled by clinical observation.

Tachycardia of gastric origin is so rare that we do not remember to have seen a case. The pulse may be frequent in diseases of the stomach accompanied by fever or by an inflammatory complication, and a weak heart may be excited by normal or by pathological digestion. But the normal heart beats no more frequently in the afebrile or simple diseases of the stomach than it does in health. Moreover, a symptomatic increase of the frequency of the pulse does not constitute tachycardia, for it is essential that the neuromuscular apparatus be disordered. Arrhythmia, allorrhythmia, and asymmetry are equally as rare as gastric tachycardia, though they may occur in association with other disturbances of the heart of undoubted gastric origin. It may be possible for a disease of the stomach to produce these disturbances in

either their paroxysmal or habitual forms, but it would seem wise to ignore the possibility until better proof of their causation is given than we have been able to find. Gastric bradycardia, however, is the most common form of the slow heart. The heart-beats sometimes fall as low as 35 or 40 to the minute, but the milder form with 50 or 60 beats a minute is most frequent. The bradycardia may occur in paroxysms and be accompanied by weak action of the heart, as shown by a small pulse, pallor, cold extremities, fainting, and by loss of consciousness in severe cases; it may be intermittent, the paroxysm occurring in connection with gastric digestion or with an exacerbation of gastric irritation; or it may become chronic and habitual. It is sometimes observed in neurasthenia gastrica, but it is most common in myasthenia and obstructive retention, in ulcer, in the painful paroxysms of hypersthenic gastritis, and in gastroptosis, particularly when the gastroptosis is accompanied by retention, by neurasthenia, and by low abdominal tension.

Nervous palpitation may be the expression of a disease of the stomach, and, indeed, in either its subjective or its objective form. The subjective form is characterized by normal heart action, the complaints of the patient being due to hyperesthesia of the sensory nerves of the heart. This pseudopalpitation occurs sometimes in ulcer, adenohypersthenia gastrica, chronic hypersthenic gastritis, and gastralgia, the epigastric cutaneous nerves being at the same time hyperesthetic. Objective palpitation is characterized by perceptible overaction of the heart, the frequency of the heart-beats being commonly increased. The palpitation may have in the disease of the stomach its all-sufficient cause or only the occasion of the attack. The diagnosis of the cause of palpitation may present almost insuperable difficulties, but the source of the trouble may be located in the stomach by exclusion of the diseases of the heart and blood-vessels, and of other diseases and habits (morphinism, alcoholism, abuse of tobacco, excesses in venery, etc.) which may produce it, by the close relation of the attacks to gastric digestion, and by its control or cure under treatment of the disease of the stomach with which it is associated.

Gastrocardiovascular Symptom-groups.—A well-defined cardiovascular symptom-group of gastric origin is sometimes met with in neurasthenic or nervous patients, between the ages of twenty and forty years, who suffer from bulimia, adenohypersthenia gastrica, myasthenia gastrica, or neurasthenia gastrica. The trouble is always paroxysmal in its

first stages. The attacks, which begin and end suddenly, last from a few hours to two or three days. Usually, during the night or soon after rising in the morning, a sense of oppression and fullness is felt in the epigastrium, the heart palpitates, the pulse becomes irregular, and the patient is suddenly seized with great anxiety. The heart feels overdistended and flutters, and the abdominal aorta palpitates strongly. The patient is weak and depressed, cardiac dyspnea is marked, but there is no precordial pain and no headache. The attacks recur after varying intervals, or the trouble may become continuous and chronic, with constant epigastric distention, dyspnea, bulimia, and anxiety which is likely to produce hypochrondriasis. The disease affects almost exclusively men, and chiefly brain-workers, the attacks recurring after eating an acid or some particular fruit or food. The whole trouble seems to be produced by a reflex from the morbid mucous membrane of the stomach affecting the vagosympathetic, and probably also the vasomotor, nerves. In the severe attacks the arterioles are constricted and the left heart is dilated. During the intervals between the attacks the heart and circulation are normal. The cardiovascular paroxysms can be controlled by the proper treatment of the disease of the stomach.

Another well-defined gastrocardiovascular symptom-group, produced by a disease of the stomach, affects the arterioles of the lesser circulation and causes dilatation of the right side of the heart (Potain). These attacks are more common in neurasthenic and chlorotic girls than in men, and they may or may not be painful. The attacks are sometimes brought on by very mild gastric excitants, even solid food, like digestible meats, being sufficient to produce them. The attacks occur during gastric digestion and begin with slight dyspnea and substernal oppression. In the beginning of the attack the second pulmonary sound is accentuated and has a quick, metallic ring. Later, the heart sounds become muffled, and a distinct *bruit de galop* can be heard to the right of the sternum. In the severe attacks relative tricuspid insufficiency may develop, a systolic murmur being heard at the apex, propagated to the right and accompanied by a systolic distention of the right jugular vein. The heart dulness is then enlarged to the right. The attack may end in half an hour, and may or may not be accompanied by moderately severe pain extending over the thorax from the left clavicle to the umbilicus. The trouble is most common in hyperesthesia gastrica, neurasthenia gastrica, during the development of gastroptosis, and in adenohypersthenia gastrica.

The influence of the diseases of the stomach on the heart and blood-vessels should not be forgotten, for the gastro-cardiovascular troubles can be cured only by the proper treatment of the disease of the stomach. An acute disease of the stomach or an exacerbation of a chronic disease of the stomach may break compensation, or give the death-stroke in organic disease of the heart, or be the exciting cause of an attack of angina pectoris. It is a good rule to watch the stomach in the management of the diseases of the heart and blood-vessels.

VI. INFLUENCE ON THE NERVOUS SYSTEM.

During normal gastric digestion the nervous system is physiologically in repose. It is a natural period of mental and physical rest. The inactivity may be prevented by the use of stimulants, like tea, coffee, alcohol, tobacco, and by a lively environment. But if the mind be not forced into activity, it will seek its physiological repose, and if it be too much excited artificially, the functions of the stomach may be slowly performed. In an analogous manner, pathological digestion may destroy this natural tendency of the mind and body, and the nervous system itself may manifest the disordered digestion. Gastric headache, drowsiness, insomnia, and the many nervous symptoms of the diseases of the stomach, display the influence of the diseased stomach on the nervous system. But not only are nervous symptoms produced, but also special disorders of the nervous system. The principal disturbances of this kind are neurasthenia, vertigo, tetany, and epileptiform convulsions.

There is no question in our mind that both spinal and cerebral **neurasthenia** may result from the diseases of the stomach. There is no doubt that the reverse is equally true; that neurasthenia may begin in other organs or in the central nervous system and extend to the stomach. Irritable nerve weakness may readily be propagated from one division of the sympathetic system to another. Then are established the neurasthenic vicious circles of the stomach, and the stomach itself may forge this circular chain. It matters not whether the irritable weakness be caused by self-poisoning, by subnutrition, by oligocythemia (gastric), by direct propagation along the sympathetic or the pneumogastric nerves, or by the effect of the gastric trouble on the mind or on sleep. The stomach is still the creator of the trouble. The diseases of the stomach

which are most active in this respect are neurasthenia gastrica, gastroptosis, myasthenia gastrica (with hyperesthesia, hyperchlorhydria, or fermentation), obstruction of the pylorus, and all the hypersthenic painful affections of the stomach. The disease of the stomach is the primary trouble, and the secondary neurasthenia can be cured only after the control or cure of the exciting causative disease. Like other secondary diseases, the neurasthenia may acquire an independent existence, and it always requires treatment in itself. But this peculiarity is no evidence against its genesis by the disease of the stomach.

Vertigo a stomacho laeso (Trousseau) is not frequent. Gastric vertigo is in itself without characteristic features, but it occurs in association with the stomach trouble and is relieved by the cure of the stomach disease. It is sometimes possible to bring it on by sudden change of position, and to relieve it by giving a few mouthfuls of food; it is sometimes associated with nausea, sometimes with frontal headache, sometimes with vasomotor disturbances, and sometimes with hot flushes and a sense of warmth in the stomach. The attacks begin sometimes when the stomach is empty, and sometimes a few hours after a meal. The head first feels light, or heavy, or compressed, the vision becomes cloudy, there is some particular sensation referable to the stomach, and then the surrounding objects oscillate and turn about the patient, or the patient loses his sense of equilibrium and of space and feels himself in the air. Consciousness, however, is never lost, and the patient always knows that the movements are mere illusions. Vertigo is a very common symptom, and it is a symptom of many other diseases (particularly of the arteries and the circulation) besides those of the stomach. In the cases of vertigo in which we have been able to find no other cause than the disease of the stomach, the digestion has always been "slow and laborious" (Trousseau), and the myasthenia has been accompanied by butyric acid fermentation. But we are not prepared to state that gastric vertigo may not occur under other circumstances.

Tetany is a rare complication of the diseases of the stomach, and we have been able to find only 41 reported cases. But gastric tetany is frequently fatal (73 per cent.), and many cases doubtless occur without being recognized or without being reported.

Tetany is a motor neurosis characterized by bilateral paroxysmal tonic spasms affecting chiefly the flexor muscles of the extremities. The muscular cramps are painful, and con-

sciousness is never (true tetany) or very seldom (sometimes in gastric tetany) lost. The mind remains clear (true tetany) or there may be some confusion of the intellect and a treacherous memory (sometimes in gastric tetany). There is no fever unless there be a febrile complication, and the attacks, which last for from five to twenty minutes, or possibly several hours, begin and end suddenly, and are preceded and followed by sensory disturbances, like formication, over the region of the affected muscles. After intermissions the paroxysms may recur, and the trouble may last for several days or weeks.

Gastric tetany occurs in a mild and in a severe form. In the mild form the cramps affect the muscles of the extremities, sometimes of the upper extremities only, and are confined chiefly to the flexor muscles of the forearms and hands and to the corresponding muscles of the legs and feet. The extensor muscles are also affected, and, while yielding to the stronger flexors, aid in holding the hand rigid and immovable through the influence of the will. A characteristic deformity of the hands was described by Trousseau as the "obstetrician's hand." The thumb is strongly adducted, the straight fingers are drawn against one another and are half flexed over the thumb at the metacarpophalangeal joint, and the sides of the palm are turned in to form a cone. The index-finger may be flexed to a greater extent than the others, or the thumb alone may be contractured. Frequently, however, the hand assumes the same form as in posthemiplegic contracture. The toes are contracted in an analogous manner, and the foot is commonly in the equinus position. The hands are flexed on the wrist, the arms and legs at the elbows and knees, and the upper arms and thighs may be fixed in strong adduction; but these contractures may not occur in the mild form. In the severe form the cramps begin in the extremities and extend to the thorax and abdomen; they may sometimes affect the diaphragm, the muscles of the neck, of the face, of the eyes, of the tongue, of the pharynx, and of the larynx. The pulse is rapid, and the patient may lose his life by suffocation.

The tendon reflexes are normal, or are not changed in a particular manner, but the skin reflexes are exaggerated. Compression of the main nerve of the extremity (median, sciatic) or compression of the main artery or vein of the extremity, increases the spasm during the stage of contracture and excites an attack during the intermission. It is curious to note that the effect of the compression is reflected to the corresponding extremity (Trousseau). The galvanic excitability

of the motor, and sometimes of the sensory, nerves (except the facial nerve) during the continuation of the trouble is always greatly increased, and usually the faradic current produces the same effect as the galvanic current (Erb). The mechanical irritability of the nerves is increased (Trousseau), and Chvostek discovered that it is possible to produce contraction of the facial muscles by tapping over the facial nerve or by stroking the skin and muscles of the face from above downward, along a line extending from a point midway between the eye and ear to the middle of the horizontal branch of the lower jaw. These signs of Trousseau, of Erb, and of Chvostek are present in only a part of the cases of gastric tetany, and their absence constitutes an important variation from true tetany.

Gastric tetany and tetany-like cramps result from a very limited number of the diseases of the stomach. And, indeed, when these attacks do occur, they can not be considered a result of the primary disease of the stomach, but of a special secondary condition, which represents an episode in the development of the primary disease. It is neither ulcer, nor obstruction of the pylorus, nor gastroptosis, nor myasthenia, nor chronic hypersthenic gastritis which directly cause the attacks, but it is the gastric retention that is the essential condition. In the majority of the cases the gastric retention is associated with excessive secretion or with a highly acid condition of the gastric juice. In a small percentage of the cases the retention is accompanied by active fermentation and by the absence of free hydrochloric acid. The retention need not be continuous, for it may be absent before and after the attacks; but whether the retention be continuous, or only temporary or accidental, it is a condition essential to the occurrence of the attacks. The continuity of secretion is the effect of the retention; the hydrochloric acidity of the contents is a manifestation of the diseased mucous membrane; and the organic acidity is the result of the retention and of the quality of the diet. The frequency of butyric fermentation in these cases is noteworthy and suggestive. The immediate exciting cause of the attacks may be a vaginal examination, or palpation of the stomach, or the introduction of the tube, or lavage, or the sudden evacuation of the contents of the stomach through the tube or by vomiting, or the attacks may be caused by other forms of mechanical irritation. The attacks may be produced by auto-intoxication, or, as some contend, by reflexes from the stomach, or by the extreme poverty of the tissues (muscle and nerve) in water.

It seems probable that the reflexes and the desiccation are only contributing causes.

Kussmaul may be considered the father of the desiccation theory, but he has recently abandoned it. There is no question that as a result of the retention of the excessive secretion, of the diminished absorption, and of the vomiting the system is deprived of much water. As a consequence there is oligemia sicca, very dry skin, hard feces, and a small quantity of urine, but the blood is not thickened, as is sometimes stated. Indeed, it is poor in albumin, poor in sodium chlorid, and the number of corpuscles in the cubic millimeter is diminished,—certainly not increased,—and there may be signs of excessive destruction of the red corpuscles, and of degeneration and decrease of the number of white corpuscles. But the rectal administration of water or of salt solution does not prevent or control the attacks; water starvation and excessive water elimination do not produce them, and the attacks do not occur in relation to the poverty of the system in water. It is easily conceivable how the desiccation and the changes of the blood may stimulate the absorption of the poisons which may be in the stomach, may favor their formation in the body, and may lead to their accumulation in the system by insufficient elimination.

There is little evidence of the production of these muscular cramps by reflex action. Mechanical irritation may occasion them, but just as readily when other parts of the body are irritated as when the irritation acts directly on the mucous membrane or on the wall of the stomach. Tetany, due to intestinal worms, is more likely toxic than reflex. Reflex action is the cloak in which ignorance loves to hide itself. The theory of reflex action naturally excites distrust, and there seems to be no clinical nor experimental evidence in its favor.

It seems probable that the attacks of tetany and of tetany-like cramps are due to self-poisoning. Bouveret and Fleiner have extracted from the gastric contents toxins capable of producing convulsions and death of the animal, but the convulsions were clonic. The extract of Bouveret and Devic has been shown to be a mixture containing a yellow substance capable of producing mydriasis and vasoconstriction, but it is found, like the ethylendiamin of Kulneff, in cases with no convulsions of any kind. It has also been shown by Fleiner that the alcoholic extract is poisonous when the neutralized contents are not toxic. Bouveret and Devic suggest that the poison may be formed in the stomach by the digestive action of free HCl in the presence of alcohol (swallowed or gener-

ated by fermentation). It has also been suggested that the poison may be formed by prolonged gastric digestion (peptotoxin) and may be absorbed slowly and continuously and without special alteration by the diseased or healthy mucous membrane. Ewald and Jacobson extracted the picrate of an alkaloid-like body from the urine of a tetany patient, and Albu found a double gold and platinum alkaloidal salt in the urine during the attack which was absent from the urine in the interval. Consequently, we may conclude that there is no chemical nor experimental evidence in favor of the intoxication theory, for a poison capable of producing the tetany-like cramps has not been found in the stomach contents before and during the attacks, nor in the blood during the attacks, nor in the urine during and after the attacks. These failures exclude the probability of acute self-poisoning, but do not refute the hypothesis of a slow cumulative poisoning. The evidence in favor of the auto-intoxication theory is chiefly clinical. The cramp-producing agent acts like an alkaloidal poison and produces no anatomical lesion except nephritis (toxic) in some cases. Tetany, so far as at present known, is always toxic, occurring in the course of or during the subsidence of, infectious diseases, after the extirpation of the thyroid gland (mucin-toxemia), after the administration of certain chemicals or drugs, and, sometimes, during pregnancy or lactation. And gastric and tetany-like cramps occur in close causal relation with gastric retention, the gastric contents containing a mixed and active virulent germ growth.

The *convulsions* which result from a disease of the stomach may be *epileptiform*. After a sensation of weight or of painful dragging of the stomach, of nausea, or of regurgitation, general tonic, followed by clonic, convulsions begin, and are accompanied by loss of consciousness. The gastric epileptiform attacks should be distinguished from true epilepsy with a gastric aura, and they may occur as the only convulsive manifestation of the gastric disease, or they may occur in a patient who has attacks of tetany-like cramps. Explanations of the production of the cortical irritation are hypothetical, and the prognosis is better than in true epilepsy and in gastric tetany.

VII. INFLUENCE ON THE SKIN.

Through the influence of the diseases of the stomach on the vasomotor nerves and on nutrition, and through the irri-

tation produced by the elimination of the products of self-poisoning, the skin may become diseased. No doubt intestinal diseases are more active in the causation of diseases of the skin than are the diseases of the stomach, but in some cases the stomach is the sole source of the skin trouble. Urticaria is popularly supposed to be due to a bad stomach. A fugacious form often results from the eating of berries or shell-fish, and it is difficult to determine whether this angioneurosis is produced by reflex action or by self-poisoning. The fugacious and chronic forms are most common in children, as a result of the acute or chronic myasthenia induced by overfeeding. Urticaria may also result from gastric fermentation in the adult, but, in our observation, the urticaria is accompanied in nearly all cases of gastro-intestinal origin by intestinal putrefaction and indicanuria.

Eczema is very rare as a result of a disease of the stomach alone, but it may be excited by acid self-poisoning (fermentation) in patients predisposed to it, and the excessive acidity of the system may be the cause of the obstinacy of certain cases to treatment. We have noticed that eczema seborrhœicum is nearly always associated with butyric acid fermentation in the stomach or the formation of β-oxybutyric acid in the body. The "red nose" (rosacea) is sometimes due to myasthenic fermentation and to the diseases accompanied by hyperchlorhydria. The influence of the diseases of the stomach in the causation of the diseases of the skin has not been thoroughly studied, and it presents an opportunity for the employment of modern methods in the correction of errors and in the extension of our knowledge.

VIII. INFLUENCE ON THE KIDNEYS.

The diseases of the stomach modify directly the composition of the urine. In retention the total quantity of the urine may be reduced to one-fifth of the normal quantity. Its acidity may be increased (diminished gastric secretion) or it may be decreased (excessive secretion). There may be phosphaturia, or there may be precipitation of uric acid, or the toxicity of the urine may be increased. Functional albuminuria is frequently caused by the diseases of the stomach which produce supersecretion and phosphaturia. And, finally, the urine may contain gastric ferments and albumoses. But it is doubtful whether these changes do much direct damage

to the kidneys by their local action alone, with the exception, possibly, of the unassimilable albumin.

But indirectly, by their influence on the liver, on the blood, on the cardiovascular system, and on the nourishment of the body, the diseases of the stomach may initiate changes in the glomeruli and the tubules. The process may be degenerative, congestive, or inflammatory. Chronic degeneration may result from subnutrition in conjunction with auto-intoxication. The epithelium lining the cortex tubes is swollen and infiltrated with granular matter and fat. There is little albumin in the urine, and only a few casts (there being no changes in the blood-vessels or in arterial tension, no hypertrophy of the left ventricle, and no uremia), the patient's strength and nutrition gradually or progressively failing. But besides the chronic degeneration, an exudative or even productive inflammation of the glomeruli or of the cortex tubes may result indirectly from the diseases of the stomach. The diseases of the stomach undoubtedly influence the evolution of chronic nephritis, and a therapeutic rule may be drawn from this clinical fact. But the part which the diseases of the stomach play in the causation of the diseases of the kidneys is not definitely known, and it is very likely that their pathogenic influence may easily be exaggerated.

CHAPTER II.

THE SECONDARY DISEASES OF THE STOMACH.

THE secondary diseases of the stomach are produced by the diseases of a large number of other organs, and, as a rule, but not always, bear no marks which would reveal or suggest their origin. When once established, they are capable of an independent existence, and may have their usual evolution. They may, however, be more obstinate than is ordinarily the case, and their cure requires the cure or control of the causative disease or its natural advance to another stage in which it has not the same influence on the stomach.

The disease of the stomach may be an accidental association, developing before the beginning or during the course of the disease of the other organ, and as the effect of the same or of totally different causes. They exist together, but the one is not produced by the other.

In order that the disease of the stomach be considered secondary, it must be connected in its origin and evolution with a primary disease. What precedes can not be a result. Its course must be influenced by the primary disease, and it must be observed to follow the primary disease with sufficient frequency to be considered an order of sequence. Naturally, the probability of the etiological relation is greater when an explanation can be given of the mode of genesis.

In order to throw light on the often obscure relation, it is necessary to know the age and nature of the stomach trouble with more exactness than can be learned from the clinical history, from the physical signs, and from autopsies. Diseases of the stomach are too often latent, are too often ill-defined or similar in their subjective manifestations, too often escape a physical search, too often leave no traces perceptible after death, and too often change their nature during their terminal period, to have their age and nature revealed without the use of the modern methods of examination.

I. DISEASES OF THE INTESTINES.

Clinical observation establishes the fact that a disease (except obstruction) of one of the divisions of the digestive tube affects the divisions of the alimentary tract below it much more frequently than those above it. Consequently, secondary disease of the stomach does not often result from intestinal disease, although the stomach is not exempt from intestinal pathogenic influences.

Symptomatic disturbances—loss of appetite, nausea, vomiting, excessive or diminished secretion—of the stomach are very common in diseases of the intestines. Active abdominal plethora may result from the active hyperemia of intestinal irritation or inflammation. Active plethora is as frequent as portal congestion, and in this manner intestinal disease may produce congestion of the stomach or gastritis.

Duodenal obstruction or chronic stenosis in the upper intestinal tract may produce gastric retention and secondary dilated hypertrophy of the stomach. If the obstruction is below the opening of the common duct, there is regular reflux of bile and of pancreatic juice into the stomach, and there is accompanying hyperchlorhydria or hypochlorhydria according to the functional power of the gastric glands. In intestinal obstruction there is antiperistalsis and excessive gastro-

intestinal secretion above the obstruction, with nausea, vomiting, and hydrothionemia.

Intestinal auto-intoxication may produce paroxysmal or digestive adenohypersthenia gastrica. The paroxysmal attacks may be very severe—nausea, vomiting, cramps, severe headache, anxiety, depression of spirits, collapse, and even stupor. The stools are very foul, there are H_2S flatus and indicanuria, and sometimes H_2S can be detected in the breath and in the urine. Chronic intestinal auto-intoxication may produce chronic gastritis by its direct and indirect influences.

Intestinal neurasthenia and the hypersthenic chronic affections of the intestines may produce neurasthenia gastrica, and duodenitis may extend by continuity of structure to the gastric mucous membrane. Enteroptosis, also, may produce gastroptosis, but in our opinion the particular pathological influence of enteroptosis has been somewhat exaggerated by Glénard. The enteroptosis is as frequently the result as it is the cause of the displacements of the other abdominal organs, and all these displacements are more frequently the result of a common cause—emaciation and lack of proper support.

II. DISEASES OF THE LIVER.

In the diseases of the liver secondary diseases of the stomach are common and have not received the study and recognition which their frequency and clinical importance demand.

The chemistry of digestion may be disturbed by the reflux of bile into the stomach, which may excite excessive secretion, nausea, and vomiting. In all our cases of chronic reflux of bile into the stomach there has been constant hyperchlorhydria, and such is probably the rule, unless there be antecedent asthenic gastritis or gastric atrophy.

Simple enlargement of the liver may produce vertical displacement of the stomach and interfere with the performance of its motor work. Obstructive stagnation is not seldom produced in this manner, the evacuation of the stomach having been delayed in a little more than half of our cases, accompanied by noteworthy enlargement of the liver from various causes.

In catarrhal icterus and in infectious cholangitis hyperchlorhydria is the rule, and in only a few of our cases have we found secretion normal. But we have studied too few of

these cases to do more than emphasize the desirability of further investigations.

Hypertrophic cirrhosis produces hyperchlorhydria with more or less stagnation (13 of 18 cases). The chronic hypersthenic gastritis (9 of 18) may have been due to the same cause as the hypertrophic cirrhosis, but it is important, at least, to remember the frequency of the association. In severe cases of atrophic cirrhosis we have always found hypochlorhydria, the result probably of portal congestion or of chronic gastritis.

It is often stated that hyperchlorhydria is an important differential sign between gastric ulcer and cholelithiasis. We have found hyperchlorhydria in 17 of 23 cases of gall-stones, and 3 of the 6 remaining ones had chronic asthenic gastritis. Hyperchlorhydria occurs as frequently in cholelithiasis as in ulcer, but in 5 of the 17 cases it disappeared in the interval between the attacks. The hyperchlorhydria is cured by the passage or removal of the stone, and its persistence after an attack of gall-stone colic should excite suspicion of stones still remaining in the ducts or in the gall-bladder.

Pyloric or duodenal stenosis may result from carcinoma of the gall-bladder or from gall-stones. The inflammatory complications of gall-stones may produce cicatricial obstruction, but the pylorus may also be obstructed by the presence of a gall stone in the common duct. If, under such circumstances, there be gastric retention, pain, vomiting, and supersecretion, ulcer with pyloric obstruction would probably be the diagnosis erroneously made. Or if there be pain, vomiting, gastric stagnation or retention, emaciation, and absence of free HCl, carcinoma would be suspected, and the erroneous suspicion would become a belief if, as often happens, a tumor (the stone) should be felt in the pyloric region.

III. DISEASES OF THE HEART AND ARTERIES.

So long as the valvular diseases of the heart are compensated the functions of the stomach are not disturbed by them in any manner, and the heart may become moderately or temporarily insufficient without producing an appreciable or more than evanescent diminution of secretion. But in the asystolic stage of chronic valvular disease the passive congestion of the stomach causes secretion to become insufficient or the free hydrochloric acid may entirely disappear

from the contents obtained after the test-breakfast. If gastritis has not resulted from the congestion of the stomach, the secretion will become normal after the integrity of the circulation is restored by proper medication. If gastritis, on the other hand, has been produced, the gastric secretion will be permanently under the influence of the anatomical changes of the mucous membrane. After cyanosis and dropsy are well established, no medication, in our experience, restores the lost or impaired secretion. The examination in 18 cases during the period of compensation revealed no disease of the stomach nor disturbance of secretion which could not be readily explained by the action of other causes than the disease of the heart. In compensated heart disease gas often accumulates in the stomach, although secretion be normal and no signs of fermentation can be detected. Seven of these 18 cases complained greatly of the flatulency. Twenty-three cases examined when the signs of heart insufficiency were marked showed normal secretion (5), diminished secretion (13), and the complete absence of free HCl (5). The ferments do not diminish so rapidly as the free HCl, and in none of the cases of valvular disease examined by us during the stage of broken compensation have we found hyperchlorhydria or myasthenia.

Arteriosclerosis and cardiosclerosis, or aortic valvular disease secondary to chronic disease of the arteries, produce three forms of stomach trouble. In eleven cases we found the digestion of the test-breakfast normal, but there was no free HCl after the Riegel test-dinner, albumin digestion being proportionately defective. The stomach is capable of doing a small task, but soon becomes exhausted if prolonged work is required of it. Again, we have observed six cases of arteriosclerosis which presented, from time to time, a peculiar form of gastralgia or gastrospasm. These attacks occur during the digestion of a meal (usually large and somewhat excitant in its physiological action), and resemble the muscle cramps of arteriosclerosis. It is probable that they are due to anemia, produced by the gastric localization of the arteriosclerosis, and are not associated in any regular manner with disturbances of secretion. Again, arteriosclerosis may produce chronic interstitial and atrophic gastritis (eight cases), and it is a noteworthy fact that this form of secondary gastritis is sometimes accompanied by motor insufficiency. The relative secretory insufficiency, the digestive gastrospasm, and the mixed gastritis may develop at different periods during the course of the arterial disease. In only three of the cases of

well-marked arteriosclerosis thoroughly studied by us have we found the stomach normal, but in some of the cases (which have been excluded from consideration) the chronic gastritis was in all probability due to the medication or to the advanced nephritis.

IV. DISEASES OF THE BLOOD.

The diseases of the white corpuscles (pathological leukocytosis, leukemia, and leukopenia) do not produce any special disturbances of the stomach. The enlarged spleen which accompanies leukemia may be the cause of vomiting. The enlarged liver may obstruct the venous circulation of the stomach, and interfere with the proper performance of its mechanical work. Leukemia may, however, produce symptomatic gastric hemorrhage. Leukopenia and leukocytosis do not affect the stomach, although either functional or organic disease of the stomach may result from the same cause which produces the leukopenia or the leukocytosis. The symptomatic gastric hemorrhage of scorbutus and of hemophilia need only be mentioned.

The diseases of the red corpuscles are responsible for the secondary diseases of the stomach which are due to the diseases of the blood. But not all the gastric troubles which are found in the anemias are due to the disease of the blood. The stomach disease and the blood disease may be accidental associations, or they may result from pathogenic causes which affect both, or the gastric trouble may be the effect of the medication employed against the anemia, as the iron, arsenic, and the excitant diet which it is the rule to prescribe. Eliminating, so far as possible, these disturbing factors, our investigations have led to the following conclusions:

Simple oligochromemia disturbs neither secretion nor the motor function. But this is not the case in chlorosis. In about one-third of the cases of true chlorosis the stomach symptoms predominate in the clinical history; in about one-half of the cases there are gastric symptoms and pain. Vomiting occurs intermittently in about ten per cent. of the cases, and loss of appetite is the rule. Disturbances of secretion are more frequent than would be indicated by the complaints of the patients, as in only nine per cent. of the cases have we found secretion normal in its degree and in its evolution. Gastric secretion is sometimes normal when the patient complains of digestive trouble.

As regards the nature of the stomach trouble, in 23 per

cent. of the cases we have noted a diminution of secretion—in some cases functional, in other cases due to gastritis. But we can not convince ourselves that the gastritis is due to the chlorosis and not to other causes. In 68 per cent. of the cases we have found the hydrochloric acidity excessive at some period during the digestion of the test-breakfast—excessive free HCl at the expiration of one hour in 11 per cent. of the cases, excessive physiological HCl (H + C) in 32 per cent. of total cases, and an abnormality in the evolution of digestion in the remainder of the cases. No noteworthy myasthenia existed in any of the cases, the results being obtained by using the ordinary methods and by controlling them with the water-test whenever they did not give satisfactory results. Briefly, in chlorosis there may be hyperesthesia gastrica, or there may be adenohypersthenia gastrica, and in some cases there is hypersthenic gastritis. Ulcer was present in six per cent. of the total cases, and we have never seen a gastric hemorrhage in pure chlorosis that was not due to ulcer. Gastric cramps occur in chlorosis when it is accompanied by gastroptosis or hyperchlorhydria. Associated with the gastric trouble is generally found neurasthenia gastrica, which is as it should be when the influence of chronic irritation or irritable weakness of the abdominal sympathetic in the causation of chlorosis is held in mind.

The disturbing influence of oligocythemia on the stomach is far less than the influence of chlorosis. Hemorrhagic anemia diminishes secretion, and this effect is often seen in ulcer after a severe hemorrhage. But the normal secretion or the hyperchlorhydria returns as the regeneration of the blood advances. In grave oligocythemia secretion may be diminished, and the gastric glands, like the noble elements of other organs, may undergo fatty degeneration and atrophy. Ulcer is much less frequently a result of oligocythemia than of chlorosis. In the study of the effect on the stomach of oligocythemia, be it dyshematopoietic, degenerative, or hematocytolytic, we have been unable to discover a thread to guide us in the confusion. In the primary cases it is difficult to eliminate the influence of medication or to estimate the influence of the causative disease in the secondary cases, as in pyemia, in septicemia, and in the anemias due to intestinal putrefaction and auto-intoxication. In a majority of the simple cases of mild and severe oligocythemia the stomach is normal.

V. DISEASES OF NUTRITION.

The various diseases of nutrition do not disturb the stomach to an equal degree. Chronic rheumatism produces directly no particular disturbance. Obesity causes little more than diminution of the appetite, the gastric disturbances found in this disorder of nutrition being due to the mode of life and to improper treatment. But many fat people are active, enjoy good appetites, and have excellent digestion. Fasting produces diminution of hydrochloric acid secretion without diminishing the formation of the ferments. The stomach ceases to act, but retains its functional power. In chronic subnutrition there is diminution or loss of HCl secretion without a corresponding diminution of the ferments. But in prolonged subnutrition of a severe degree secretion is not simply in abeyance, but it may be permanently impaired, and the weakness of the stomach muscle is in keeping with the weakness of the general muscular system. We would, in this connection, emphasize the very important influence of emaciation and loss of muscular strength in the causation of displacements of the abdominal viscera—kidneys, liver, spleen, colon, and stomach.

Diabetes, be it constitutional (nutritive), nervous, pancreatic, alimentary, or hepatic, may disturb the stomach. In many cases there is only a diminution of HCl secretion. There is often myasthenia in cases of long standing, which may be associated with hyperchlorhydria or with hypochlorhydria, but which is most likely the result of excessive eating and drinking rather than of the diabetes. It is the rule to find no serious disturbance of the stomach in diabetes, unless there be great emaciation, advanced cardioarteriosclerosis, or nephritis. But it should be remembered that the functions of the stomach may be insufficient, and under such circumstances the diet should not be made too exclusively nitrogenous.

The gastric troubles of gout may be due to the drugs which are commonly employed, to the restricted diet, to the secondary nephritis and arteriosclerosis, or, finally, to the disease of which the uric acid precipitation is the expression. There may be an associated or, sometimes, a secondary gastritis. But the special gastric trouble of gout is myasthenia, which may be accompanied either by hyperchlorhydria or by hypochlorhydria, with or without fermentation. If there be hypochlorhydria and fermentation, a vicious circle is established, for this secondary gastric trouble of gout favors

the conversion of the neutral into the acid phosphate of soda, and may cause uric acid precipitation.

VI. DISEASES OF THE KIDNEYS.

It is difficult to study the effects of the diseases of the kidneys on the stomach, for the usual medication of nephritis is likely to do the stomach injury, and the two organs may become diseased from the same causes. But excluding, so far as possible, these sources of error, it may be stated in a general way that the stomach troubles of nephritis are due to acute or chronic uremia. The retention poisoning may act on the central nervous system and produce vomiting, which is always a most prominent gastric symptom in uremia, whether gastritis be present or absent. The retention poisoning also leads to the elimination of ammonia compounds by the stomach, and the HCl may be neutralized, so that the analysis of the contents gives a false conception of the activity of secretion. We have frequently noticed that the hypochlorhydria of the acute exacerbations of chronic nephritis is replaced by normal secretion, or even by hyperchlorhydria, during the period of quiescence of the Bright's disease when renal sufficiency is reëstablished. Indeed, it seems that it is the rule in the early period of chronic nephritis to find the gastric irritation displayed by hydrochloric acid in excess. But later, the hyperchlorhydria is replaced by permanent hypochlorhydria symptomatic of chronic gastritis. The hydrochloric acidity diminishes during the uremic attacks, and the alkaline or nearly neutral vomit may contain ammonia (white cloud produced by vapor from a glass rod dipped in HCl). The ferments seem to be destroyed in part, or are secreted in less quantity than would be proportionate to the diminution of the hydrochloric acid secretion. Flatulency is common, although fermentation is rare, and it may possibly be due to the decomposition of carbonate of ammonia. The stomach disturbance is a rough index of the degree of renal insufficiency, and the preservation of the functions of the digestive organs protects the system and the kidneys against injury by gastro-intestinal auto-intoxication.

Stone in the kidney may either produce no gastric trouble at all or it may excite reflex vomiting. We have sometimes found hyperchlorhydria, or, more frequently, hypochlorhydria. The painful gastroduodenal crises of movable or floating kidney are said to be common, but the disturbance certainly

originates in some cases in the cecum and colon, and in others the signs and symptoms are due to perinephritis.

VII. SPINAL DISEASES.

Myelitis, multiple sclerosis, and spinal meningitis may be accompanied by reflex vomiting, by hyperchlorhydria, and by painful gastric crises. But the gastric troubles caused in this manner are either so rare or so obviously sympathetic that they hardly deserve mention. It is not so, however, with the gastric crises of locomotor ataxia, which occur during the course of the sclerosis of the posterior columns, or which may be the first revealing sign (in about five per cent. of cases) of tabes at a period when there are no disturbances of the reflexes, of sensation, or of coördination.

The gastric crises begin suddenly, regardless of the state of repose or of functional activity of the stomach, and regardless of the quantity and the quality of the diet. There may be irregular prodromal symptoms—shooting pains, epigastric uneasiness, depression of spirits, and restlessness. The crisis is continuous, and is manifested by pain, by vomiting, and by general weakness and anxiety. In from a few hours to several days the crisis ends as suddenly and as apparently without cause as it began.

The pain is not always present, and it is variable in quality and intensity. It may be burning, stabbing, shooting, cramp-like, moderately severe, or almost deadly in its agony. But the pain has always certain distinctive characteristics: it is bilateral in its radiations; it is not relieved by vomiting, by alkalies, or by albuminous food; it is only temporarily diminished by lavage, and then only in the beginning of the crisis, and morphin controls it only during the period of narcotism. Sometimes, though seldom, the pain is the only manifestation, and it may then be cramp-like, without vomiting, and with complete arrest of secretion. Furthermore, we would emphasize the fact that the quality and the intensity of the pain bear no relation whatever to the hydrochloric acidity of the contents of the stomach.

Vomiting may be absent, but usually it is present, obstinate, and accompanied by nausea and by retching. It may be the predominant symptom, and the gastric intolerance may be complete. The vomit consists of whatever may be in the stomach at the time—food, gastric juice, mucus, and, eventually, bile and pancreatic juice. The crises are usually accompanied by thirst and by complete loss of appetite.

The crises are not always of the same severity, and consequently do not always produce the same effects on the general system. The vomiting and pain may be so severe that complete collapse is produced by the uncontrollable vomiting and the intolerable agony. As a rule, the crises become milder as the spinal disease advances.

The crises of tabes dorsalis are paroxysmal, spasmodic, and gastralgic. The nerves and the muscle of the stomach are affected to a much greater degree than is secretion. There is pain, loss of appetite, and cramps, but the state of secretion is determined to a greater extent by the antecedent state of the mucous membrane than it is by the spinal sclerosis. If there be no disease of the mucous membrane, in the beginning of the crisis there is hyperchlorhydria; but in case the crisis is prolonged or is repeated after short intervals, the vomit contains less and less HCl and ferments. Under such circumstances gastric secretion is normal during the intervals. But it is the rule, as a result of the antisyphilitic or antitabetic medication, to find chronic asthenic gastritis already present. The quantity of hydrochloric acid in the vomit in the beginning of the attack displays the secretory power of the diseased mucous membrane, and the acid is consequently diminished. During the course of the attack the acidity becomes less, and the diminution is due in part to the reflux of the duodenal contents into the stomach, and to the capillary hemorrhage which occurs in some of the very severe attacks. There seems never to be continuous secretion, nor is there myasthenia with retention or accumulation of the secretion of the stomach. Medication has no appreciable effect in the control or prevention of the crisis. It will be seen at a glance how different are the functional signs of the gastric crises of locomotor ataxia from those of chronic hypersthenic gastritis or from the paroxysmal form of hyperchylia gastrica, the two diseases with which the gastric crises are most likely to be confounded.

VIII. CEREBRAL DISEASES.

Meningitis,—particularly basilar meningitis,—cerebral hemorrhages, brain abscess, and brain tumor, may be manifested by vomiting. The vomiting, which has the peculiar characteristics of cerebral vomiting, is central and symptomatic, and can not be properly considered a secondary affection of the stomach. Cerebral vomiting is easy, projectile, without rela-

tion to meals or to the quality of the diet, is not accompanied by disease of the stomach, and may occur in crises. In every case of obstinate vomiting it should be a rule to look for possible cerebral lesions and their other manifestations—vertigo, headache, motor and sensory disturbances, and changes of the optic disc and retina. Brain injury has been known to produce capillary gastric hemorrhage. Hyperchlorhydria is more frequent in melancholia, in dementia paralytica, in mania, and in paranoia than is normal secretion, though hypochlorhydria is also found. Cerebral fatigue may produce paroxysmal hyperchylia gastrica, but the most common gastric affection which originates in the central nervous system is neurasthenia gastrica.

It is very difficult to define and explain the gastric troubles of neurasthenia. It may be contended that the disease of the stomach is only an accidental association, be it a dynamic affection or an anatomical disease. It may be contended that the gastric trouble is primary, and that it is the cause of the central neurasthenia. All the hypersthenic diseases of the stomach may produce general neurasthenia, and in latent forms may, during a variable length of time, be manifested by weakness and increased irritability of the central nervous system, even without subjective gastric symptoms. It may be contended, and sometimes with truth, that the central neurasthenia is primary, and that it may produce functional disturbances of the stomach, which in their normal evolution may become transformed into anatomical diseases. It seems wisest, in the prevailing confusion, to avoid partizan theories. If we have found in our experience that about one-third of the cases of central neurasthenia develop secondary affections of the stomach, and that about four-fifths of the cases of neurasthenia gastrica (primary) develop some of the signs and symptoms of cerebral and spinal neurasthenia, the observation may be imputed to the supposed tendency of each specialist to exaggerate the importance and the pathogenic influence of the organs whose diseases it is his particular business to treat. This personal equation may be very great, and we admit its influence; but we should be more inclined to accept the neurologist's opinion if it were more frequently the custom to examine the stomach by the modern methods. In our experience, the most common secondary disease of the stomach in cerebrospinal neurasthenia is neurasthenia gastrica. There may be no abnormality of secretion or of the motor function—the patients simply suffer and complain, and are overanxious as a result of the sensations which accompany digestive activity,

just as in primary neurasthenia gastrica. At times there may be hyperchlorhydria—free HCl may appear too early in the course of the digestion of the test-breakfast, or it may be too great at the height of its digestion, or the appearance of free HCl may be delayed and, eventually, become excessive in quantity. More infrequently there may be hypochlorhydria. Or there may be myasthenia with a mild degree of stagnation, the stomach doing its motor work perfectly for a certain period, and then becoming insufficient, as may be clearly shown by the water-test. But the functional abnormality is intermittent and inconstant, for if the central neurasthenia be accompanied by a secretory disorder of a fixed chemical type, an organic change in the mucous membrane is present. The anatomical disease may be an accidental association, or it may have developed in consequence of the predisposition created by the neurasthenia and by the neurasthenic functional disorder.

The secondary affections of the stomach produced by hysteria are more numerous than those of neurasthenia. The most important are anorexia, hyperesthesia gastrica, anesthesia gastrica, and hysterical vomiting. These hysterical gastric troubles may be the only manifestations of the hysteria, or the psychosis may affect some of the voluntary and conscious functions of the organism. The monosymptomatic gastric form may present great difficulty in diagnosis. Although hysterical stigmata may be absent, the gastric affection may possess some peculiar and suggestive characteristic. The anorexia may be an anorexia with a fixed idea or purpose. The vomiting is always alimentary and easy. In spite of both anorexia and vomiting, nutrition may be perfectly preserved for a long time, and there is no organic disease of the stomach present to cause them. These affections begin suddenly and without an apparent cause, and end suddenly and in an inexplicable manner. They have no relation to the quality or quantity of the diet, and often run their course irregularly and in a manner typical of no particular disease of the stomach, and in total disregard of all, except suggestive treatment. These and many other well-known hysterical characteristics may suggest the nature of the puzzling and grotesque affection.

IX. DISEASES OF THE MOUTH, NOSE, AND THROAT.

It is exceedingly difficult to determine the relation of the diseases of the mouth, nose, and throat to the diseases of the

stomach. In the first chapter of the Vicious Circles of the Stomach no mention was made of the possible causation of diseases of the upper air-passages by the diseases of the stomach, because such a result seems to be very infrequent. Acid regurgitation may affect the throat as it does the esophagus, and the diseases of the stomach may influence the circulation, the nutrition, the nervous system, etc., in such a manner as to predispose more or less all the mucous membranes of the body to inflammation. That is about as much as can safely be said without danger of exaggeration. The autotoxic sore throats due to H_2S poisoning are of intestinal origin.

Diseases of the mouth may disturb gastric digestion by interfering with mastication or by changing the reaction and composition of the mixed secretion of the mouth; and thrush may, under fit circumstances, extend from the mouth to the stomach.

It is often contended that the frequent coexistence of nose and throat disease with gastric disease demonstrates that one or the other disease is secondary. But the two may be produced by the same cause, or they may be the effects of different causes. It is hard to find a perfectly normal nose and throat in New York, but diseases of the stomach are by no means so frequent, and in fine climates and in better hygienic surroundings the two classes of diseases are far less frequently associated.

Diseases of the nose and throat may influence the stomach by reflex action, by local irritation, by infection, or, in some cases, by the influence on the general health. After tabulating a large number of cases, we are unable to detect the reality of the reflex pathogenic influence on the stomach. There is no disturbance of secretion or of the motor function, which appears with regularity in connection with diseases of the rhinopharynx. The swallowing of large quantities of mucopus for a long period does affect the stomach, and the pyogenic cocci may grow on the mucous membrane if there be but little HCl secreted; such, anyway, would seem to be the case, as we have seen pyogenic cocci in large numbers on little pieces of the mucous membrane found in the expressed gastric contents. In chronic asthenic gastritis, associated with purulent rhinopharyngeal diseases, the micro-organisms of the stomach are a close reproduction of the micro-organisms which are swallowed. The tabulated cases show another noteworthy fact—the diseases of the stomach which are most frequent are associated with the most frequent diseases of the

rhinopharynx. While the etiological relation of the diseases of the stomach and the diseases of the throat, nose, and its accessory sinuses is still an open question, there can be no doubt that it should be the rule of practice to treat both diseases simultaneously.

X. DISEASES OF THE RESPIRATORY ORGANS.

Acute pleurisy produces no particular disturbance of the stomach, for the visceral congestion of its initial period is but slight, and the fever does not run high. Large effusions may interfere with the aëration and circulation of the blood, and with the movements of the diaphragm, which facilitate the circulation in the abdomen by subjecting the organs to rhythmical compression. But gastric digestion proceeds in a normal manner if proper attention be given to the diet, though reflex vomiting sometimes occurs, and the pleuritic pain, as does all severe pain, may depress the functions of the stomach. The disturbance of gastric digestion, if it occurs, passes away in a short time without doing any particular damage. Such is not the case, however, with empyema, for the toxemia depresses secretion, induces myasthenia, and sometimes leaves chronic asthenic gastritis as a legacy.

Acute bronchitis affects the stomach to a greater degree than does acute pleurisy. It appears that acute gastric catarrh with hyperchlorhydria, which is the common disturbance of the stomach in acute bronchitis, is only an associated disease due to a common cause. Chronic bronchitis and emphysema produce chronic defective aëration of the blood and dilatation of the right side of the heart. Passive congestion and catarrh of the stomach may result, and the swallowing of the expectoration of putrid bronchitis may disturb secretion and irritate the mucous membrane.

Pneumonia affects the stomach very seriously, and lobar pneumonia is more active in this respect than is the lobular form. Obstinate reflex vomiting may occur from the irritation of the pneumogastric by the compression of the consolidated lung, and it often follows the cough. The prolonged chill, high fever, defective aëration of the blood, and the insufficiency of the right side of the heart produce the most common disease of the stomach in pneumonia, which is acute gastritis. The gastritis may develop during the onset of the pneumonia, but it often appears later, when the heart insufficiency and cyanosis are most marked. The initial gastritis is accompanied by hyperchlorhydria (vomit of 17 cases), but the

terminal gastritis is accompanied by hypochlorhydria (vomit of three cases) or by absence of free HCl (vomit of eight cases). Fortunately, neither pleurisy, bronchitis, nor pneumonia produce myasthenia. The motor function remains unimpaired, and the stomach empties itself normally throughout these diseases, unless it be maltreated or already diseased.

Of all the grave organic diseases, pulmonary tuberculosis exerts the greatest influence on the stomach, and thus weakens or destroys the system's defense against the progress of the disease. If nutrition is to be maintained and fortified, —and all agree that this is the essential basis of antitubercular medication,—the integrity of digestion must be preserved. If the drug treatment is to be beneficial, the recuperative and resisting powers of the body must be increased. As soon as the functions of the stomach are lost, the lungs and the whole system are at the mercy of the tuberculosis.

Not all the gastric troubles which occur in the course of consumption are due to the tuberculosis. The stomach may be already diseased when the tubercular infection begins, and, indeed, gastritis (Hayem) and "dilatation" (Bouchard) create favorable conditions for the development of pulmonary tuberculosis. Consequently the gastric trouble may be primary and not secondary. And, again, during the course of the tuberculosis the gastric trouble may be produced by ordinary causes, and not only in spite of, but sometimes as the result of, the medication adopted. The stomach is neglected and injured in the absorbing effort to control or cure the pulmonary disease. Or the stomach may be affected indirectly by the influence of the pulmonary tuberculosis (mixed infection) on nutrition and on the blood. But, apart from all these possibilities, we maintain that pulmonary tuberculosis frequently exerts a direct and injurious influence on the stomach.

Our conclusions are based on the study of 95 cases. Of these, 47 were first seen during the incipient period, 26 during the stage of consolidation, and 12 during the period of cavity formation. In every case the diagnosis was confirmed by demonstrating the presence of the tubercle bacillus, which was not found in seven of the cases of incipient tuberculosis until several weeks after the patient first came under observation, these cases beginning clinically with circumscribed dry pleurisy of the apex of the lung.

Of the 47 cases of incipient tuberculosis, ten manifested no subjective nor objective signs of an anatomical disease or of a dynamic affection of the stomach. Three of the remaining cases had chronic asthenic gastritis; there were

absence of free HCl, diminished combined HCl, corresponding diminution of the ferments, excessive quantity of mucus, and no (or very slight) impairment of the motor function. Eighteen other cases gave only traces of free HCl, diminished combined HCl, slight diminution of ferments, with mild (13) and severe (5) forms of stagnation associated with more or less active fermentation. It is more than probable that the gastric trouble in these 18 cases antedated the tuberculosis, but the motor insufficiency was, probably, the effect of the tuberculosis, as primary mild asthenic gastritis of the ordinary type is characterized, with but few exceptions, by preservation of the motor function. Retaining the myasthenia as the only tubercular characteristic, these 18 cases may be excluded, along with the three cases of chronic asthenic gastritis, from the study of the gastric troubles of the incipient period. We will return later to the discussion of the evolution of these cases, eight of which complained of only irregular disturbance of the stomach and three seemed perfectly satisfied with their digestion. The 16 remaining cases of this group will now be reviewed. Three of them suffered from the digestive form of adenohypersthenia gastrica; there was free HCl in large excess, diminished or normal combined HCl, excess of ferments, no excess of mucus, and a perfectly preserved motor function. Seven other cases had myasthenia with stagnation of the first degree, associated with secretory irritation; they showed free HCl in excess, and delayed evacuation of the stomach. One of these patients had the characteristic pain and tender points of ulcer, and gave the history of hematemesis, there being no blood coloration of the sputum during the following day. The six remaining cases gave the same functional signs, but fermentation was also present in all of them, and in two of the cases the stomach succeeded in emptying itself only during the night. In these last two cases the free HCl dropped below normal after the stomach was thoroughly washed out and a proper diet had been given to control the fermentation. Briefly, incipient tuberculosis produces simple digestive adenohypersthenia gastrica in about six per cent. of the cases, and myasthenia, with or without fermentation, leading on slowly or rapidly to gastritis, in about 28 per cent. of the cases. If the stomach be already diseased, it becomes myasthenic.

Of the 26 cases first examined during the stage of consolidation, in only four was the stomach normal. Five were cases of chronic asthenic gastritis with severe stagnation (4),

or with retention (1), and with no free HCl (5). It was not possible to determine whether the gastritis was secondary to the tuberculosis in all these five cases. Of the remaining 17 cases of this group, three displayed excessive hydrochloric secretion, 13 variable (sometimes normal, sometimes excessive, sometimes diminished) HCl secretion, and one gave only a trace of free HCl. Of all 17 cases, myasthenia with fermentation was present in the mild (5) or severe (10) stagnation or in the retention (2) form. Consequently, during the stage of consolidation there may be myasthenia with secretory irritation, or there may be gastritis with motor insufficiency.

All the 12 cases first examined during the stage of cavity formation proved to be cases of chronic asthenic gastritis with motor insufficiency. In four of these there was gastric retention, and in all of them there was gastric fermentation.

The evolution of the gastric trouble was followed closely in some of these cases. By lavage and diet the fermentation could be controlled in the cases with cavities or softening, but the functions of the stomach were not perceptibly improved. Little more could be done than adapt the diet to the obstinate trouble. Of the 17 cases in the consolidation group, 11 progressed slowly toward complete disappearance of the free hydrochloric acid, and the motor insufficiency increased or remained stationary for a short time. The fermentation was controllable, but the functions of the stomach could not be improved. The results of the treatment of the gastric troubles in incipient tuberculosis were more encouraging. Both the hyperchlorhydia and the fermentation in all 16 cases were controlled by treatment. The three cases of adenohypersthenia developed myasthenia as the pulmonary trouble progressed, and the one which lived longest nearly lost the free HCl before death. Of the remaining 13, four have completely recovered their gastric functions, and the tubercular process is stationary; three others developed gastritis before death; the remaining cases were not further studied by the modern methods of examination. The stomach troubles of incipient tuberculosis are sometimes curable, and by proper treatment the secretory irritation and the fermentation may be controlled. The development of gastritis may be postponed. Myasthenia is the most obstinate and the most characteristic gastric trouble of phthisis. The gastritis may result from the stagnation and secretory irritation, and, clinically, may manifest itself as the hypersthenic or asthenic form. Round-cell interglandular and subglandu-

lar infiltration with cloudy swelling and degeneration of the secretory cells predominates, either ultimately or from the beginning, over productive glandular inflammation and hyperplasia. The gastritis of phthisis is a mixed gastritis.

It is very interesting to follow the evolution of the 14 cases the functions of whose stomachs were normal when the patients first came under observation, ten being first seen during the incipient period and four during the stage of consolidation. Four of the ten incipient cases complained of great malaise and gastric discomfort after meals, and, being anemic and nervous, were probably suffering from neurasthenia gastrica. All ten cases became myasthenic during the stage of consolidation, and the four living several months longer developed asthenic gastritis. The others died without further exploration of the stomach, having been taken off by tubercular pneumonia. Of the four normal cases seen during the stage of consolidation, one had suffered from reflex vomiting, one displayed only mild myasthenia two months before death, and the vomit of another, examined during his last days, contained no free HCl two hours after he had taken a glass of milk. But the pre-agonal gastritis should be excluded from the study of the cases of gastritis due to pulmonary tuberculosis.

But, apart from these gastric troubles of phthisis,—adenohypersthenia gastrica, myasthenia, and gastritis,—consumptive patients sometimes present a very characteristic form of vomiting. The vomiting is probably reflex, and is a secondary dynamic affection of the stomach. The vomiting is alimentary, occurs after eating,—not after every meal (exceptions few), but chiefly after the midday or the evening meal,—begins during or after coughing, is without nausea, is performed without effort, and ceases without leaving much discomfort. This peculiar form of vomiting may be the only affection of the stomach which is present at the time, or it may be associated with the adenohypersthenia or with the myasthenia gastrica. It was noted in 17 of the incipient cases. After the development of gastritis the vomiting changes in character, and may occur without preceding cough, either after eating or when the stomach should be empty, and is accompanied by nausea and retching. If retention be present, it may be projectile, copious, and recur every two or three days, the vomit consisting in part of food which should have long ago been evacuated into the duodenum. It is easy to recognize the reflex vomiting of consumption, and its presence should always direct attention to the lungs.

The fever of pulmonary tuberculosis is without much influ-

ence on gastric secretion. The stomach seems to become accustomed to fever, and is not disturbed by the high temperature, as it is in the infectious diseases. The control of the fever by antipyretics does not improve the insufficiency of secretion in pulmonary tuberculosis. However, in the three cases of adenohypersthenia gastrica, excessive acidity disappeared when the temperature became persistently high. When the hyperchlorhydria depends upon the local irritation of the stagnant contents it does not subside as the fever rises, nor is the diminished secretion, due to asthenic gastritis, restored by the control of the fever. Indeed, the preservation of the appetite, in association with febrile movements and a rapid pulse, should direct attention to the lungs, although the patient may complain only of the stomach.

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